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HUNTERIAN LECTURES

ON

INTRA-CRANIAL INFLAMMATIONS

STARTING IN THE

TEMPORAL BONE

THEIR COMPLICATIONS AND TREATMENT

Delivered at the Royal College of Surgeons, June, 1889

BY

ARTHUR E. J. BARKER, F.R.C.S.

Hunterian Professor of Surgery and Pathology; Surgeon to University College Hospital, &c., &c.

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LECTURE I.

Mr. President and Gentlemen,—The subject to which I have the honour of inviting your attention in this and the following lectures is but a fragment of a much larger one which may be contemplated alike by the physicians and surgeons of this country with peculiar satisfaction. Whether we turn to the history of the pure pathology, of the clinical observation, or of the treatment of intra-cranial disease, we find that our countrymen have ever been foremost in the field of discovery, have cultivated it with the greatest vigour, and have won from it the largest yield of practical results.

We may well be proud of the work of such observers as Hughlings Jackson, Ferrier, Yeo, Gowers, Schæfer, and Horsley, who have been foremost in reducing the chaos of cerebral pathology to something like order; of the labours of Sir Joseph Lister, which have rendered the interior of the skull as safely accessible to us as any other cavity in the body; and of the operative achievements of his followers, who, like Macewen, Godlee, Horsley, and others, have been privileged to gather the first-fruits in the great harvest-field of modern brain surgery.

But much, of course, remains to be done on this ground in various directions; and to one particular branch of the subject in which I have been greatly interested, and in the study of which I

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have for the last twelve years enjoyed some exceptional advantages, I will now ask your attention, as it appears to me to demand a far broader treatment than it has yet received. I refer to the inflammatory changes within the cranium and their complications, due to disease of the temporal bone.

The careful study of this branch of the great subject of cerebral pathology and surgery is surely of the utmost practical importance, for several reasons.

In the first place, whatever may be the ultimate verdict of science as to the advantages of removing malignant and other tumours from the brain, there can be no question as to the desirability of relieving the cranial cavity of inflammatory products which are immediately dangerous to life. And further, a broader study of these secondary inflammatory conditions within the skull by the profession at large is sure to result in a more thorough and widespread recognition of the dangers of the primary affections within the temporal bone which give rise to them, and in earlier and more determined efforts in the direction of prophylaxis. It will probably be a very long time before any means are discovered for preventing the growth of neoplasms within the skull, but it must be perfectly evident to any one who gives a patient attention to the inflammatory intra-cranial complications of ear mischief that this group of diseases might soon, by more thorough heed to the primary conditions, either be stamped out altogether, or at least so far limited as to become of extreme rarity, instead of being, as now, of very common occurrence, and frequently destructive of life, to say nothing of the sense of hearing.

That suppurative disease of the temporal bone is very common every medical man of experience must be aware. But that a correct notion as to its dangers and its relative mortality to that of other diseases is entertained by the bulk of the profession, and by the public, I very much doubt. Some are inclined altogether to underrate the seriousness of suppuration of the ear, while others are equally prone to exaggerate its dangers. As a matter of fact, it is by no means easy to express the true state of the case in figures. And yet it *is of the utmost* importance that we should have something like

clear conceptions upon the point. In the endeavour to obtain these I have ransacked a great mass of home and foreign statistics of various kinds bearing upon the question of risk from many sides, and yet I cannot but feel that the figures about to be quoted have only an approximate value.

In the first place, an appeal to the Registrar-General's Reports gives us only a faint shadow of the truth. If you will glance at this table, compiled from his Reports for the last ten years, you will see that in this country between 350 and 400 individuals are stated to die annually of what is vaguely termed "otorrhœa" or "otitis," in the official record. But you may also observe, in the same Reports, a very large number of deaths set down every year to "simple meningitis;" and a much larger number still, amounting to many thousands, grouped as unclassified brain disease, and under the heading of general septic affections.

TABLE I.

REGISTRAR-GENERAL.

Deaths from "Otorrhœa," during the Years 1878 *to* 1887 *inclusive.*

	Males.	Females.	Total.
1878	151	116	267
1879	137	106	243
1880	167	115	282
1881	203	156	359
1882	255	198	453
1883	175	168	343
1884	228	163	391
1885	222	193	415
1886	214	195	409
1887	224	184	408
Total	1,976	1,594	3,570

Now, if it were possible to sift all these latter groups, and to rearrange them according to the primary cause of death, there

can be no doubt that these annual 400 deaths, attributed to ear-disease, would be swelled to four or five times the number. And I do not hesitate to say again, that this loss of life might easily be prevented by the application of that same careful antiseptic treatment to inflammatory disease within the temporal bone which is now almost universally applied to purulent affections of all the other bones of the body.

One of the causes why this class of affections is so rife is the widespread neglect by medical men generally of the study of the pathology and clinical features of the commonest ear diseases Hitherto the latter have been too frequently quietly relegated, if not actually contemptuously tossed aside, to our aurist *confrères* after delay and neglect has quadrupled the difficulties of the latter to deal with them successfully. And men who have taken pains to acquire familiarity with the laryngoscope and ophthalmoscope, and with the diseases they reveal, and also with the appearances within the rectum, vagina, or bladder, as seen through special instruments, have almost shown a pride in professing complete ignorance in regard to matters relating to the dangerous processes going on within the cavity of the temporal bone, which we call the ear, processes which are no whit more difficult of study, and which are quite as important.

This state of things must have an end sooner or later, and the sooner the better. And it will be a matter of much regret if a country which can show such admirable pathological and clinical work as that of Toynbee, Gull, and Wilde, three or four decades ago, should stand still, enjoying the credit of their labours, without any serious endeavour to promote by all means the science which they had so much at heart, and the foundations of which they laid so soundly in our midst.

Among the most dangerous ear affections, which are at the same time by a long way the commonest, the question of the patient's present and future welfare is determined by the treatment of the first few hours of the attack, and every physician and surgeon ought to be as competent to recognise such affections, and to treat them promptly, as he would be to detect and direct the treatment of an acute tonsillitis, a strangulated hernia, or an internal pile.

Even supposing that the treatment of the *suppurative* affections *alone* of the middle ear were undertaken as matters of ordinary practice ; if thoroughly studied and understood, there would be an immense gain to the public, while abundance of material, as will be shown presently, would be left on which our specialist brethren would find ample scope for the exercise of their most useful powers.

In many respects there has been for some time past a distinct improvement in this direction. In the ordinary out-patient departments of a few of our metropolitan hospitals the rich material of aural cases has been of late years utilised in such a way as fairly to familiarise the present generation of students with ear disease, *not as a speciality*, but as a matter of ordinary routine practice. But there is still much room for improvement here, and every one can contribute something to popularise this much-neglected subject. But above all, when every qualifying examination in medicine and surgery includes questions in the pathology and treatment of diseases of the ear, still greater impetus will be given in the right direction.

I have some hope, therefore, that in handling this question of intra-cranial lesions due to ear disease with no preconceived notions, and merely as a matter of ordinary study, I may perhaps help to show that familiarity with the latter is not beyond the reach of those devoted to every-day surgery like myself. My own acquaintance with these matters is due to the kindness of my colleagues, through whom nearly all the cases of serious ear disease seeking relief at University College Hospital during the last twelve years or so have passed through my hands, to the number of some thousands of individuals. This has furnished ample material for study, and I have tried to supplement it by the dissection of a large number of specimens, and by the perusal of much of the current literature of the subject. I may, perhaps, be permitted to say here that for my own part the more I learn about the morbid conditions starting in the temporal bone and their sequelæ the more I am convinced that a very thorough acquaintance with their pathology, symptoms, and treatment is necessary to those who would treat successfully any

but the most trivial of the secondary morbid conditions due to them. In other words, the physician or surgeon must not rely for his diagnosis upon the knowledge and experience of the aurist, but must acquire these for himself; for it is into his hands, in the first instance, that such serious cases are most likely to come for diagnosis and treatment; and the latter, to be safe and effectual, must be undertaken at very short notice, as a rule. Moreover, it is only by uniting a general knowledge of medicine and surgery to this thorough acquaintance with ear inflammations that very grave errors in the diagnosis and treatment of the intra-cranial sequelæ are to be avoided; so that these cases cannot be safely relegated to the hands of aurists, however much we shall always remain indebted to them for the special treatment of some of the other after-effects of ear disease.

With these few prefatory remarks your attention will first be asked to that part of our subject which, I venture to think, is most likely to be neglected at the present time—namely, *the kind of mischief* in the temporal bone which is likely to give rise to intra-cranial inflammations. We shall then consider the *modus operandi* of the production of the latter, and finally, their differential diagnosis and treatment.

When we come to look for the points at which primary inflammation arises in the temporal bone and spreads to the intra-cranial structures we find that the internal ear and external meatus may practically be left out of consideration. Primary suppurative inflammation of the *internal* ear (by which is meant the labyrinth, etc.) is admitted by all experts to be next to unknown so far. And though suppuration is frequently met with in the *external* ear as a primary and separate condition, it is very uncommon for such a process to give rise to any intra-cranial complication so long as the membrana tympani remains undestroyed by it, and the tympanic cavity uninvaded; and such an invasion of the middle ear from the external is also of very uncommon occurrence. It is, then, in the middle ear (including in this term the cavity of the tympanum, with its offshoots into the mastoid process and pars squamosa, and the Eustachian tube) that we have to look for the primary disease, the secondary effects

of which within the skull we are now considering, and which are so frequently fatal.

I venture to think that we do not sufficiently realise how far the affections of the middle ear preponderate over those of the other two sections of the auditory apparatus. And yet it is of importance that we should do so. To state this in figures it is necessary to analyse far larger numbers of cases than those which would come under the observation of any one man. And here let me remark that we are enormously indebted to those specialists in otology who have gone on for years carefully recording and classifying their cases according to one definite plan, until we have masses of statistics of the utmost value, and which could hardly be furnished by those who see these cases, no matter in how large numbers, in the course of their ordinary medical or surgical work.

TABLE II.

Diseases of the Auditory Apparatus Classified.	Bürkner, 16 different Authors.	Bezold.			Bezold's Average for 14 Years.
		1872 to 1880.	1881 to 1883.	1884 to 1886.	
External ear	25·5	24·6	24·6	23·8	24·3
Middle ear	66·9	69·2	66·5	64·4	66·7
Internal ear	7·6	6·2	8·9	11·8	8 9
Total number of cases analysed	43,730				11,654

If you will glance at this table, in which Bürkner embodies the statistics of sixteen observers, who classified their cases practically in the same way, you will see at once the relative proportion of middle ear disease to all other affections of the auditory apparatus. On a total of 43,730 cases of ear troubles of all kinds, 25·5 per cent. were affections of the external, 69·9 per cent. of the middle, and 7·6 per cent. of the internal ear. And if we turn to Bezold's analysis of his own cases for fourteen years (1872 to 1886), which

might be assumed to be of more uniform classification, the percentages are not materially different; *e.g.*, on a total of 11,654 cases the external ear shows 24·3 per cent., the middle 66·7 per cent., and the internal 8·9 per cent. of lesions. Examining some 430 consecutive cases seen by myself in private, of which the notes have been fairly accurately kept, I find the middle ear affections still more numerous, standing at 78 per cent., while the external ear only shows 16·5 per cent., and the internal only 3·8 per cent. This difference may be accidental, or, what is more likely, may be due to the greater attention to cleanliness of the external ear found among well-to-do patients; the fact, too, that these last figures are from the notes of a surgeon, and not of an aurist, would also affect the proportion of serious cases, in all probability.

Now, taking the affections of the middle ear as about 67 per cent. of all aural trouble, further examination shows that a large proportion were *suppurative* affections of the tympanic cavity. In short, of all the ear lesions classified in these records over 29 per cent. were accompanied by purulent inflammation, acute or chronic, in the very centre of the temporal bone. This is well shown in Table III.

TABLE III.

MIDDLE EAR DISEASE.

BEZOLD.				BURKNER.
1872 to 1880.	1881 to 1883.	1884 to 1886.	Average.	Average of Sixteen Authors.
Suppurative.				
26·2	28·6	32·4	29·0	29·0
Non-Suppurative.				
43·0	37·7	33·0	37·9	35·9
Number of cases analysed... ...			11,654	43,730

My own experience in the few thousand cases which have been under my care would, I believe, tally exactly with that just given,

although, from the loss of case papers and other disturbing causes connected with out-patient hospital practice, I am unable to furnish the figures in detail.

Here, then, in this 29 or 30 per cent. of all known aural lesions we find suppuration in the body of the temporal bone. Further analysis shows this to be acute in 5 per cent., chronic in 24 per cent., and it is among this last large group of affections that we must look for the most fertile sources of those intra-cranial lesions with which these lectures are more immediately concerned.

As to the broad etiology of these suppurations in the temporal bone, I need not do more than remind you that first in the list of primary existing causes stand the acute specific fevers, next the acute and subacute catarrhal affections of the naso-pharynx, and lastly traumatisms, or, suppurations spreading from the external meatus through the membrana tympani. Any of these suppurations may be accompanied by those secondary septic processes which we have learned to dread in all parts of the body, but which occurring here in close proximity to the brain and other important structures, are peculiarly dangerous.

The minute pathology of these particular putrefactive changes has within the last few years been greatly advanced by the discoveries in micro-biology which have so largely enriched o knowledge of the causation of disease in other parts of the body. And although the number of observations on the organisms present in the suppurations within the temporal bone, and their sequelæ, is so far limited, they are well deserving of notice here, as being highly suggestive and worthy of much wider study.

It seems highly probable, from the bacteriological investigations of Zaufal and others, that in the production of those forms of acute otitis media, due to cold, which are accompanied by catarrh of the naso-pharynx the pneumo-bacillus of Friedlander and the diplococcus of Frænkel play an important part. Zaufal has also shown that the streptococcus pyogenes is largely, though not exclusively, present in those dangerous and often fatal secondary complications of chronic otitis media with which we are concerned, and that its demonstration in any discharge from the ear ought to influence the prognosis most unfavourably. These

observations have been confirmed by several other observers, among whom Moos, Rohrer, and Netter may be specially mentioned. Rohrer's observations are particularly interesting from their completeness. He examined the discharges from the ears of 100 patients suffering from otitis media, both directly by stained cover-glass preparations, by cultivation, and lastly by inoculation experiments on animals. The first point noticed was a great contrast between the *fœtid* and *non-fœtid* discharges. In the former both cocci and bacilli were always found together; in the non-fœtid, on the other hand, cocci alone were present. In the offensive secretions there were about 58 per cent. of bacilli to 42 per cent. of cocci, of which half were diplococci. The odourless discharges, on the other hand, showed only cocci of which some 50 per cent. were staphylococci, 26 per cent. diplococci, 19 per cent. monococci, and 5 per cent. streptococci.

From his inoculations of animals with cultivations and pus emulsions, Rohrer came to the conclusion that the various forms of bacilli found in the *fœtid* secretions of the ear were *not* pathogenic, but simply saprophytic, the animals inoculated with the bacilli either in the tympanic cavity, the auricular veins, or the peritoneum being alive and well at the end of some months, little or no reaction having taken place locally. But of the pathogenic nature of the cocci there could be no doubt, from his experiments on animals, typical septic diseases of various kinds being produced without fail.

These observations appear to me to possess a special interest as regards the question of fœtor from the ear. It has been commonly taught hitherto that a bad smell from the ear is an important factor in the prognosis of aural inflammations. My own observations, however, for a long time past have led me to question this conclusion very seriously, and to hold and teach that some of the most dangerous sequelæ of otitis media may be met with where the secretions from the tympanum are either nearly or quite odourless. If this be true, and I fully believe it to be so, the explanation is found in Rohrer's observations regarding the pathogenic cocci found alone in the non-fœtid discharges, and the preponderance of merely saprophytic bacilli in the fœtid. We must not, therefore, think the less seriously of a discharge from

the ear because it is odourless, but must endeavour to get rid of its exciting cause just as strenuously as if it were most offensive. This is only what we might expect from an experience of ordinary suppurating wounded surfaces in other parts, which in many cases give rise to serious or fatal septic complications without giving off any fœtor.

It appears probable also, as suggested by Zaufal, that hereafter cultivations from ear discharges will afford considerable help in the prognosis of this class of diseases.

Moreover, another fact of great significance has been brought out by bacteriological study, namely, that those peculiar collections of inspissated pus in the middle ear which are familiar to all who have given much attention to the pathology of this region, and which are known to our German *confrères* as cholesteatomata, are more largely infested with dangerous micrococci than any other collection in the cavities of the temporal bone. And in this we have the best explanation of the great frequency of fatal complications where this material is present. The necessity of thoroughly removing it either through the meatus or, what is far better, by freely opening up the mastoid cavities with a gouge from behind, is emphasised by what we have learned of the specific character of the virus it contains. Here is no question of mild irrigation, but of thorough extirpation as of a malignant new growth, and this must be done with a due regard to the fact that our operation upon an infecting focus must be carefully planned, and carried out *with proper precautions against reinfecting the very wound we have made for its removal.*

Some observations which I have been conducting within the last few months in reference to these points are not sufficiently complete to justify the putting them forward in evidence as to the nature of the organisms concerned in the production of suppurative middle ear disease. But as far as my cultivations go they strengthen my belief in the accuracy of Rohrer's observations as to the saprophytic nature of the bacilli found in the discharges, as contrasted with the pathogenic properties of the several forms of micrococci present. In these cultivations with pus from the middle ear I have found large numbers of bacilli in those colonies derived from discharges which were very fœtid, but otherwise not

troublesome, while various forms of micrococci were present in all kinds of discharges, especially the more acute.

Netter, who has conducted an important series of independent observations upon the same subject, recognises four different pathogenic factors in the production of the *primary acute* attack :—

1. The streptococcus pyogenes.
2. The staphylococcus pyogenes.
3. The pneumococcus of Frænkel.
4. The pneumobacillus of Friedlander.

He is thus in most points in harmony with those observers already quoted.

In working at this aspect of the subject one is tempted to speculate as to whether this variety in the pathogenic organisms of intra-tympanic inflammation may not fully or in part account for the differences in the intra-cranial sequelæ; whether, for instance, one organism may not produce the acute non-fœtid cerebral abscess, another (possibly in company with a saprophytic bacillus) the more chronic fœtid abscess of the brain. Again, diffuse septic meningitis may be produced, perhaps, by one organism which finds its most congenial soil on serous membranes, localised plastic meningitis by another less virulent, and pyæmia by a third, just as we see variety in infecting processes in other parts of the body.

I have also thought that it would be most interesting and useful if we could undertake a series of inoculations of the brains of animals with pure cultivations of the various organisms bred from the ear. We might in this way be able to clear up some of the very difficult questions which crop up in the clinical study of these diseases. For instance, it might be made evident why the fœtid abscesses of the brain have a great tendency to lie latent for a long time. This might be shown to be due to the abscess having been caused by the presence of some of the mainly saprophytic bacilli, having little tendency to produce widely destructive local change, and having no general infective properties. An explanation might also be found here for that tendency which such fœtid abscesses possess to reduce the temperature below the normal standard, as will be shown presently. This might be found to be

due to the action of the decomposition products of simply saprogenic organisms upon the heat centres through the blood without any further pathogenic effect. This lowering of temperature is in striking contrast to the elevation which accompanies the various forms of meningitis, and can hardly be due to any pressure effects alone, which must be far greater in the latter diseases. It is also, we know, occasionally observed in certain more or less chronic intra-abdominal inflammations, and the parallelism might be explained by the presence of similar organisms there. Possibly also the decomposition products of the various organisms might be isolated from the bacilli themselves in a pure form, and their different effects upon temperature, pulse, and respiration might be demonstrated by careful injection into the brain or arachnoid space or the general circulation. This would entail an amount of labour not to be realised by any one who has not worked in this field experimentally, and could only be settled by the combined efforts of many observers skilled in bacteriological investigation. I cannot help feeling, however, that it is in this direction that the key to a proper understanding of these intra-cranial complications of ear disease will be found, and I have some hopes of testing the accuracy of these conclusions before long.

Now, if intra-cranial inflammations due to ear disease depend upon the presence of pathogenic organisms, it is of course of the utmost importance to ascertain in what way or ways they gain access to the interior of the skull.

That the path they travel upon is, as a rule, a more or less direct one is proved by the fact that auro-intra-cranial disease is almost invariably upon the same side as the affected ear, and that we can frequently roughly trace the inflammatory process along a definite course from the ear to the contents of the skull.

But in other cases the mode of inoculation of the intra-cranial structures with the *materies morbi* is not so evident, and is still, indeed, hypothetical.

Let us consider now, in the first place, those modes of propagation of the ear disease inwards which have been actually observed, and then those which may be inferred with more or less certainty. In this consideration I have not merely had to rely

upon scattered published records, but have been able to draw upon abundant pathological material, which I have been collecting for a long time past, and to which a more particular reference will be made presently.

Before entering upon this matter the first fact to which attention should be drawn is this, namely, that *primary acute* ear inflammation is rarely followed by intra-cranial complications. This is the experience of all who have given close attention to the subject, and my own observation supports it. The explanation is probably this, that as long as the mucous membrane lining the tympanum and its accessory cavities is undestroyed, and the underlying bone is unaffected, the disease is not propagated inwards. It appears probable that in such a case the lymphatics of the mucous membrane, as long as they are intact, are adequate to carry off the deleterious products of the inflammation before their local effects can reach deeper. For this reason we often find in primary acute attacks that the neighbouring glands are for a time swollen and tender, while the temperature is raised, and other signs, more or less serious, of septic absorption are present. I believe that a very large amount of septic matter may be thus taken up, carried into the circulation, and either destroyed there or eliminated by the usual emunctories.

But with chronic ear disease the condition of things is quite different. Here, instead of a lining of mucous membrane and periosteum, the walls of the tympanum consist of bare bone, perhaps more or less carious, and covered by granulations, which in themselves are easily destroyed by active septic processes. Now, these bony walls contain arteries and veins in abundance, which communicate with those of the dura mater. Either kind of vessel may obviously become the channel of infection for the latter. And from what we can see with the naked eye in the larger veins, it is plain that a septic phlebitis plays a large part in several of the morbid processes with which we are concerned. But what we see in the larger veins may be inferred to take place in those which are too small for anything short of microscopic examination. There can be no doubt that in this way sapræmia may be produced by the access to the blood simply of the products of putrid changes in

the middle ear in sufficient quantity to cause grave illness, but of a kind easily recovered from when the source of putridity is cleansed. In the same way pathogenic organisms themselves may be received into the circulation in sufficient quantity to produce its total contamination or secondary colonies in distant parts, resulting in the first case in septicæmia, and in the second in typical embolic pyæmia. Or, if the dose be moderate, and the plastic processes be sufficiently vigorous to throw out a limiting mass of exudation at the point of inoculation, a thrombosis of a sinus or a localised subdural abscess is the result. If the infection be not thus limited the organisms gain access to the serous surface of the dura mater, and set up a more or less severe lepto-meningitis or encephalitis by direct contact. Again, in chronic middle ear disease septic matter will often make its way into the labyrinth, and the infection spreads from thence along the nerves, through the internal auditory meatus, to the meninges, or to the encephalon itself.

All these modes of propagation of septic inflammation have been known for a long time past, and any one who has given much attention to post-mortem studies must have seen instances of each. But the discovery of the pathogenic properties of certain organisms has not only rendered these modes of origin of intra-cranial inflammation more easily intelligible, but has suggested explanations of certain anomalies hitherto observed in the relation of ear disease to the contents of the skull. Among these may be mentioned, for instance, the fact that in many of these cases the tympanic disease has existed for long periods without producing any intra-cranial mischief, and has then at last suddenly become dangerous on the occurrence of some septic catarrhal attack elsewhere, such as pneumonia or pharyngitis due to foul air. Indeed, it seems the rule that disease of the tympanum *which has once become chronic requires to be engrafted afresh by an acute attack before it becomes dangerous.* Its proneness thus to be freshly inoculated is of course a matter of common observation. There seems good reason to believe that in such cases the original inflammation of the middle ear had, so to speak, spent itself in destroying the mucous lining of the cavity and a certain amount of bone, but had then lost all its specific characters, and

subsided into a solely saprophytic process; but on the infection of the air passages or of the discharging meatus with the pathogenic organism the Eustachian tube and tympanum, having participated in the inflammation, became a second time the seat of a special inflammatory process. The latter, under the circumstances, would find a soil ready made for the propagation of the new organisms, and would find in the diseased vessels of the inflamed bone an easy route to the intra-cranial structures. The discovery of Friedlander's pneumobacillus and Frænkel's diplococcus in the ear in acute catarrhal attacks, as already hinted above, would suggest this sequence of events strongly to my mind, taken together with facts established by clinical observation, and especially that it is not the primary acute catarrhal process that we have to fear, nor yet the chronic inflammation developing out of it; but it is the engrafting upon the latter of a fresh inoculation of pathogenic germs that bears with it the terrible risk the fatal issue of which we so frequently see.

There is another fact regarding the sudden onset of grave complications, superadded to chronic ear disease which has given little or no trouble perhaps for years, to which I should like briefly to draw attention. We may often notice that patients who have had a flux from the ear for a long time, and who have sought surgical advice, perhaps, on account merely of the discomfort of the thing, *become suddenly very ill immediately after measures have been adopted for clearing out the middle ear* of dried up *débris* or granulation polypi. In such cases the temperature will rise high, there will be malaise, anorexia, sometimes a rigor, and all the signs of septic absorption. More than once death from septicæmia or pyæmia has been observed after such interference with old foci of disease in the ear. I cannot say that I have ever myself seen an *actually fatal* result from such operative interference, but I have often observed patients to become suddenly very ill indeed for some days, with high fever, etc., where a focus of chronic disease had been interfered with. In one or two instances, also, the sudden development of patches of *pneumonia*, with a high temperature, cough, etc., immediately

after such an operation, has been most suggestive. Cases of this kind have led me to speculate whether my own manipulations on a septic cavity close to some of the largest vessels in the body had not actually set free some of the dangerous material into the latter by the breaking down of plastic barriers which had been erected around it. The poison in such a case would, of course, be carried directly to the lung. Experience of this kind has made me very cautious about operating at first sight upon cases of chronic caries of the tympanum. Of late a course of careful antiseptic treatment has always, where possible, been made the prelude to any operative interference except of the mildest kind. And at the present day, when this class of disease is receiving so much more attention than formerly, and operations are becoming more common, I venture to think that caution in the direction indicated cannot be too earnestly pleaded for. We should be exceedingly careful lest operative interference with caries of the tympanum, which ought to be so beneficial, obtain a bad name from being adopted over hastily, and without first minimising the risks by every means.

Occurrences like these just alluded to may, I think, be explained in two ways at least. In the first place, we usually find in these cases a larger or smaller amount of stiff cheesy-looking *débris* in the middle ear, which is nothing but the inspissated products of the chronic catarrhal process. Now, this material has been found to swarm with pathogenic organisms, but in its half-dried fatty substance it may well be that the conditions do not exist for the full development of their noxious qualities. Let this material, however, be disturbed by a partial removal, or let it be moistened with water or soaked with blood or serum after the removal of granulations in its neighbourhood, and the most favourable conditions for the development of the septic cocci are produced; and though they may have been dormant for a long period, they may now multiply to a dangerous extent. But it is quite possible also that a more direct infection of the blood-stream may be produced where such half-dried foci of septic matter are disturbed by operative interference. If the latter be too vigorous or unskilful, it is quite possible that small

veins about the middle ear, previously sealed by chronic plastic inflammation, may be opened up mechanically, and the organisms may pass directly into them in large quantity, just as a torn vein may be infected by broken up caseous tubercular material during an operation. And I believe that this may occur even in the most skilful hands. These are probably the two commonest modes of infection of the system, due to interference with the ear in the course of chronic inflammatory disease. But of course there is always, besides, the risk, as in other operations, of inoculating the inflamed surfaces with pathogenic or saprophytic matter from without, and which did not previously exist in the ear, by means of instruments or fluids used for injection; but from what I have seen myself, I should regard this as a rare cause of those serious complications which often follow interference with old disease of the tympanum.

Again, bacteriology, I think, helps us much in understanding those very anomalous cases in which ear disease gives rise to a deep-seated abscess in the brain or cerebellum, *while the surfaces of the convolutions are to all appearances quite healthy.* Such cases have given rise to much speculation hitherto. For my own part I have always thought that they could generally be explained on anatomical grounds up to a certain point, beyond which there was considerable difficulty. I had often seen and demonstrated small veins passing from the external surface of the temporal lobe either to the superior petrosal sinus or to the dura mater about the roof of the tympanum. The same are found occasionally running from the anterior surface of the cerebellum to the lateral sinus. The presence of these vessels, which are not so fully described as they ought to be in ordinary literature, offers the readiest explanation of the propagation of disease from the ear direct to the substance of the brain without any evidence that the serous surfaces have been affected. What most commonly occurs is probably as follows: A septic phlebitis is set up in the venous radicles running from the surface of the inflamed tympanum to the lateral and petrosal sinuses, then thrombosis takes place at the seat of inflammation. From this *moment on there* are obviously two ways in which pathogenic

organisms can be rapidly conveyed to the brain along a radicle running into the vein thus affected. In the first place the thrombosis may extend steadily backwards contrary to the current, and so reach the cerebral tissues, carrying with it colonies of organisms. On the other hand, if one of these veins be simply blocked at its distal end its current is reversed, and the blood flowing back through it from an inflamed tissue will bear with it septic products which on reaching the brain or cerebellum set up the same process beneath the surface there. The fact that intra-cranial veins and sinuses are destitute of valves renders such a reflux easy. The first mode of propagation of inflammation to the brain, which I described some years ago, is probably the commonest form; but, indeed, both these forms have been recognised and treated of long before that, first by Dr. Peacock in the Pathological Transactions, and again by Dr. Adams, in the *Glasgow Medical Journal*, papers which, I am sorry to say, I have not read until quite recently. The small veins running from the surfaces of the brain to the roof of the tympanum, as described, may also be inflamed, of course, without any implication of the sinuses, and may thus originate uncomplicated cerebral abscess.

Now, to any one who has watched the cultivation of septic organisms it must be obvious that a very slight reflux, or the extension of a very small thrombus backwards from a putrid process in the middle ear, will be quite sufficient to set up a similar process in the substance of the encephalon, without affecting the surface of the latter. And the exact nature of that process will depend upon the species of germ imported. We have long been aware that disease in the ear may produce either a diffuse, an acute, or a well-defined chronic abscess in the brain, and such a collection of pus may be intensely fœtid, as in the first case operated on by myself, or totally inodorous, as in my second case.

These differences can be best accounted for by the variety of the organisms which originate the disease in the ear. Some of these, as observed by Rohrer, are capable of setting up acute septic processes without generating any fœtor, while others, notably the bacilli, appear to be entirely saprophytic, and to produce localised suppuration with intense foulness, without any general

pathogenic properties. And arguing from Rohrer's observations, and from some cultivation experiments which I have made myself, not yet complete, I think it not unlikely that before long it may be actually shown that the localised fœtid cerebral abscesses are originated by organisms possessing little more than saprogenic properties, or, at all events, that they are peopled by a preponderance of these; while the diffused acute encephalitis, so often accompanied by meningitis and by symptoms of general septic infection, will be demonstrated to be originated by a totally different class of organisms. This is only what we might infer from facts already known regarding local and general infection elsewhere; but when the details of the different processes have been worked out experimentally on the brain, as I hope they will be before long, they may be expected to furnish us with many useful data for prophylaxis, for prognosis, and for treatment.

Again, it must not be forgotten that the arteries passing close to the walls of the tympanum may also, as well as the veins, be the carriers of pathogenic substances from an inflamed surface to the intra-cranial structures.

As far as the dura mater is concerned, it is not difficult to understand this, inasmuch as there is a free inosculation of the arterioles of the ear and the dura through the thin walls of the former. If, then, an arteritis is produced in any of these small vessels, due to the septic processes around it, morbid products may be carried into the vessels of the dura, and start the same disease in it. Or thrombi may form at the affected spot, and breaking off give rise to emboli, which will originate inflammation at those spots in the arterioles of the dura at which they jam.

But what occurs in the smaller vessels of the tympanum may also take place in the larger trunks. Thus the internal carotid artery may be extensively diseased in that part of its course in which it is only separated from the carious tympanum by a very thin lamella of bone. This I have seen myself, and have also read of in published records. Now, from such a diseased patch in the wall of the internal carotid artery the blood-stream may be contaminated by septic products, and may carry disease to any *part of the brain.* Or a clot may form over the inflamed spot,

and give off small emboli from time to time, which will block the smaller vessels of the encephalon. This mode of propagation of ear disease to the brain will probably account for those rare cases where a cerebral abscess has followed ear disease without pyæmia, but the abscess has been found at some point remote from the affected temporal bone, as, for instance, in the frontal lobe or in the opposite hemisphere. A most interesting case of thrombosis of the internal carotid artery, as the result of intra-tympanic inflammation, is recorded by Professor Gairdner and Dr. Barr, of Glasgow, whose work in this field of pathology is particularly valuable.

While referring to disease propagated to the internal carotid artery from the ear, I should like to throw out a suggestion, which, so far as I know, has not yet been made. Attention has more than once been called to cases of tympanic suppuration, where, without any other evidence of intra-cranial inflammation, there has been more or less appearance of so-called optic neuritis or choked disc. I believe I have seen such cases myself. In speculating upon these it has occurred to me that perhaps an explanation may be found in the fact that the lymphatics, leaving the skull by the carotid foramen, may have become inflamed from proximity to the tympanic mischief. These lymphatic vessels are derived, as is well known, from affluents following the several intra-cranial branches of the internal carotid artery, and among these the ophthalmic. Now, it is conceivable that inflammation of the lymphatic trunks in the carotid canal, and their possible obliteration, might seriously retard the course of the fluid in the absorbents of the optic nerve; or a thrombosis might start in the inflamed lymphatic trunks, and extend backwards to the lymph spaces about the ophthalmic artery, so producing engorgement of the lymphatic and venous circulation of the fundus oculi.

Again, is it not possible that damage to the carotid plexus of the sympathetic from intra-aural inflammation might produce vasomotor disturbances in the optic nerve and fundus independent of any brain disease?

These are suggestions which I feel it my duty to place before

you for as much as they are worth, with all deference to, and subject to correction from, those whose labours entitle them to be considered specialists in the field to which these affections of the optic nerve more particularly belong. This point might, I think, be proved experimentally upon animals by operations upon the contents of the carotid canal, and by inoculation of them with various forms of septic matter.

But leaving these questions, I think there can be no doubt that the lymphatics play their part, as well as the veins and arteries, in propagating inflammation from the tympanum to the brain and meninges, as they certainly do in conveying septic matter from a focus in the middle ear into the general circulation.

LECTURE II.

WE must now endeavour to find out, as nearly as it is possible to do so, in what proportion of cases of suppuration in the temporal bone the septic processes may be expected to spread to the parts within the skull.

To estimate this with anything like an approach to accuracy is a matter of the greatest difficulty. It is only by examining the consecutive records of many years of work, either of special ear hospitals or of those general hospitals which have an active aural department, that this difficulty can be overcome.

But such records, to be reliable, should contain full and accurate post-mortem notes of cases dying as a consequence of disease of the temporal bone which had been recognised during life, and to mix with such cases those in which intra-cranial inflammation had been diagnosed during life, but where either recovery followed or, in the case of death, no necropsy could be made would probably lead to erroneous deductions. I have, therefore, in endeavouring to obtain information upon this point, kept the two lines of evidence quite distinct. Allusion has already been made to the yearly statistical records of Bürkner and Bezold. These bear upon them the stamp of as great accuracy as can be expected from the records of special institutions with no large command of beds in which serious cases can be followed out to the end, and where the pathological investigations may not, perhaps, be conducted upon the broadest principles. These records I have first carefully analysed, excluding such years as appeared to contain insufficient data, and grouping those in which the information was full and carefully noted. This summary furnishes us with large masses of cases recorded continuously by competent observers, and allows of as accurate deductions being made as are possible from any clinical work in which post-mortem verification of diagnosis is *not* always obtainable. And from what I have seen

in this field of surgery myself, I should feel inclined to regard the evidence thus forthcoming as of considerable value. In any case it is interesting as far as it goes.

But of far more value are the cases completed by post-mortem examination collected from the records of some of our general hospitals in this country. These will be alluded to presently, when the purely clinical material has been first dealt with.

But let us first take Bürkner's tables for the years 1881, 1882, 1883, and 1886, where the records appear open to no question as far as clinical observation is concerned. Here we find that out of a total of 3,322 cases of ear disease of all kinds, there were 864 of acute and chronic suppurations of the tympanum. Among these 11 were known to have succumbed to the sequelæ of the disease, giving a gross mortality of at least 1·2 per cent. This is probably much below the true average death-rate, inasmuch as many cases die from complications outside the notice of those otologists, who may have treated the primary disease. The causes of death are thus given: 6 died of meningitis, 2 of cerebral abscess, 1 of cerebellar abscess, 1 of sinus phlebitis, and 1 of pyæmia.

Bezold's tables for the years 1884, 1885, and 1886, which he analyses with great care, show 980 cases of acute and chronic suppuration of the middle ear, with 9 deaths, a death-rate of at least 0·9 per cent. From these two sets of figures combined it would appear that the mortality of suppurative tympanic disease, *as known to otologists*, is probably about 1 per cent.

Turning now to the general hospital records of this country, to which I have had access, and taking University College Hospital first, it appears that I and the gentlemen who have assisted me are credited with having taken notes of 2,835 cases of aural disease during the twelve years 1877 to 1888 inclusive. Now, we have seen, from the analysis of 43,730 cases of affections of the auditory apparatus alluded to in my last lecture, that 29·0 per cent. are usually cases of suppuration in the middle ear. We ought to have seen, therefore, in the same period, some 820 cases of acute and chronic otitis media purulenta. Of these the most serious would be admitted directly into the wards of the

hospital. Some cases, no doubt, passed in indirectly as ordinary medical or surgical material, but not in s ufficiently large numbers to seriously affect the above figures.

Now, after a careful examination of our medical and surgical hospital records, I find that during the period referred to *seventeen* patients died within the hospital of intra-cranial complications of ear disease. This would make the mortality of otitis media purulenta just a fraction over 2 per cent. I have not, however, included in the numbers just given my own two cases of cerebral suppuration, due to aural inflammation, saved by trephining and drainage, which would, without operation, have run an inevitably fatal course. Nor are two other cases reckoned, which had been under my care in hospital, but which died some time after they had ceased to attend, with unmistakable symptoms of cerebral disease secondary to the serious ear mischief for which they had been treated. If these four cases be added the mortality of suppurative disease of the middle ear not operated on would be swelled to something over 2½ per cent. Looking at the question as critically as possible, and in the light of all other information I have been able to collect from a great variety of sources, I think that this last figure is far more likely to represent the true risk of otitis media purulenta than the 1 per cent. derived from the clinical statistics of special otologists just given. Again, I am sure that this 2½ per cent. is not above the mark, and that this will be demonstrated when we have more accurate post-mortem records in future years.

Another inquiry now suggests itself naturally in passing. We have just seen the approximate mortality of suppurative middle-ear disease. Now, what is about the proportion which these deaths bear to deaths from other causes? In other words, what is about the mortality in any community from suppurative ear disease? This is one of the most difficult questions to answer, and the figures I now place before you do no more than suggest generally how few fatal cases we may expect to meet in our own routine work. Let us take the total experience of fatal cases in three hospitals lying close together in London, for the same number of years. This will probably give us a better general

impression of the prevalence of fatal suppurative disease of the ear than if we analysed a much larger number of cases collected from the scattered literature of the subject. I have, therefore, as a basis for considering this point generally, taken the entire experience of these diseases as far as they were fatal, or only just saved from inevitable death by operation at University Hospital, at the Middlesex Hospital, and at the Hospital for Sick Children in Great Ormond Street, for the twelve years 1877-1888 inclusive. Through the courtesy of the committees and staffs of these institutions, I have been given free access to their clinical and pathological archives, and every assistance in utilising them to the utmost, privileges which I here desire most cordially to acknowledge. These three hospitals, lying close together, may be said to serve the same population, each supplementing the other. Knowing, then, the exact number of patients who have passed through them during the years specified, we may, I think, form a tolerably fair general estimate of the amount of these diseases likely to be met with in any urban community. Again, these records have the particular merit that they are carried out on very much the same plan, and with the same general nomenclature. Moreover, the fact that the staffs of the three institutions frequently meet together at our various societies, and are thus familiarised each with the others' interests and methods of working, would tend to give a certain homogeneousness to the sum of their united records.

From the latter, then, I have compiled a careful digest of all the cases in which death was due to ear disease directly or indirectly, during twelve years. This digest, which is now before me, probably furnishes us with more accurate data for clinical and pathological deduction than could be obtained elsewhere.

It may excite some surprise, perhaps, that during the last twelve years the total number of deaths attributed to complications of ear disease occurring at these three hospitals only amounts to *forty-five*. This surprise may be increased when we learn that during the same time University College admitted 28,481 ordinary medical and surgical patients; Middlesex Hospital, 30,844; and Ormond Street Children's Hospital, 12,625; the total of

all three being 71,950 patients suffering from all varieties of disease.

Or if we take the total number of deaths from all causes occurring at the three institutions named, during the same period, we have another aspect of the case. Thus, out of an aggregate of 8,028 deaths, there were only 45 fatal cases due to disease of the temporal bone recorded.

These facts will serve in some measure to correct our notions regarding the amount of ear disease tending to a fatal issue likely to be met with, and will give us, at all events, a general impression as to its danger. Of course, some of the patients treated in these hospitals succumbed to the disease on their return home. This I know of some of my own cases. Moreover, a considerable number of patients affected with the diseases in question have been during recent years saved from inevitable death by operation at all three hospitals. These cases must also be taken into account in estimating the risks of the disease.

But it is unnecessary to pursue this inquiry further, my object having been attained in simply drawing attention to the figures above quoted. They are, I think, in a measure reassuring, in spite of the fact that probably they do not fully represent the gravity of the disease in question.

Again, this collection of 45 cases of fatal ear disease, taken from one unbroken record, enables us to work out some other points of interest and importance. It has been remarked by several very competent observers that these complications have been more frequently met by them upon the right side than on the left. Indeed, my own experience has been the same. Out of the only five cases which ultimately proved fatal under my care in private, all were, curiously enough, on the right; and in hospital right-sided disease has also preponderated among my own patients. Körner, who has made a large collection of published cases, believes that there is a distinct difference between the liability of the sides of the head to these complications, and that the left has more immunity from them than the opposite side. He believes that this is due to anatomical differences in the shape of the cranium on the two sides. From a series of observations which

he has made on a large number of skulls, he believes himself justified in concluding that the sigmoid fossa for the lateral sinus lies farther forward and to the outside on the right side of the head than on the left, and consequently nearer to the tympanic cavity, with its focus of disease. Also he believes that he can prove that in brachycephalic individuals the posterior fossa of the skull stretches farther forward on the right than in those who are dolicocephalic. Both these factors, he thinks, would explain the readier extension of the ear disease inwards on the right, especially among those with short skulls. This appears to me a matter worthy of further study, and one which might yield fruit in practice if shown to be a well-founded conclusion. But a great many points have to be taken into consideration besides the shape of the skull. In the first place, are both ears equally liable to be affected with suppurative disease? Upon this point we have abundance of thoroughly satisfactory evidence. Any large and continuous series of ear cases shows conclusively that otitis media purulenta *shows no preference* for one side over the other. Take hundreds of cases occurring in any unbroken series in any community, and the difference on the two sides will either be found immaterial or absolutely *nil*. If it be true, then, that we may expect more of these complications of middle-ear disease on the right than on the left, it cannot depend upon the initial lesion. But without invoking the anatomical explanation, I think it might possibly be accounted for by certain general habits. It may be found, on further inquiry, that more people sleep upon the left side than on the right habitually, and that consequently the right ear would be more exposed to the atmosphere, and to accidents depending thereon, than the left. Moreover, if this were so, the left ear, if suppurating, ought to drain better than the right, which would be uppermost. But upon this point, whether more individuals sleep on the left side or on the right habitually, I am not aware that we have any evidence. After all, the question which side of the head cerebral abscess, meningitis, and other complications may be expected to be found most frequently must be settled by actual observation. In our own series of 45 cases representing a continuous record of fatal disease in one community, the results are as follows: On

the right the intra-cranial complication was found in 26 cases, on the left in 22. This is not a difference in itself of any great importance, and it may be purely accidental. Indeed, it seems very likely that it is so, for if we compare the two larger hospitals we find that Middlesex, with 18 cases, has only 8 on the right against 10 on the left, while University shows only 9 on the right and 10 on the left. Thus the figures obtained from the two general hospitals for twelve years show actually more disease on the left than on the right. It is the 8 cases of right-sided disease against 2 of left-sided, obtained from the Children's Hospital in Great Ormond Street, which makes the difference in the total in favour of the right side. I confess, therefore, that Körner's view appears to me to require better proof from clinical and post-mortem statistics than he has put forward, and this in spite of the fact that by far the larger proportion of cases, fatal and non-fatal, which I have had anything to do with myself showed right-sided disease. This fact I am inclined to regard as purely accidental. In this analysis of cases it should be observed that the cases of pyæmia and meningitis are reckoned to the temporal bone on the side most diseased, from which the complication obviously took its rise.

The next point to be examined in our list is the relative frequency of the various complications starting from disease in the temporal bone. This is a most important point for practical reasons, as suggesting to us what we may expect in our clinical experience. To use an ordinary phrase, we want to know what are the odds for and against the presence of cerebral abscess, cerebellar abscess, meningitis, etc.

In the 45 cases alluded to I find that any one of the six or seven complications which may arise from ear disease rarely went alone, but was usually associated with one or more of the others. And this I believe to be the rule from the examination of a much larger number of scattered cases. The material in hand must therefore be analysed and presented to you in several different ways, to convey anything like correct impressions.

In the first place, the most prominent lesions observed were cerebral abscess in 11 cases, meningitis in 15, cerebellar abscess

in 8, pyæmia in 7, phlebitis of the lateral sinus in 3, and simple marasmus in 1. In grouping the cases in this way I have tried to arrange them according to the most striking visible lesion existing in each. Thus, we find that cerebral and cerebellar abscess make up 19 cases, meningitis and pyæmia together 22, sinus phlebitis and marasmus 4.

But if these cases are examined more closely and in the light of the clinical evidence, as well as the pathological, we find that they group themselves somewhat differently as to the apparent cause of death. There are, then, 20 which died evidently of meningitis, 14 of pyæmia, 5 of cerebellar abscess, 4 of cerebral abscess, 1 of phlebitis of the lateral sinus alone, and 1 of marasmus solely. Thus, 34 are attributable to meningitis and to pyæmia taken together, and only 9 to cerebral and cerebellar abscess alone.

From this last arrangement it would appear that the complications most to be feared in all these cases of suppuration within the temporal bone are meningitis and pyæmia. If I add to the hospital cases quoted the only fatal cases occurring in my own private practice about the exact cause of death in which I have no doubt from post-mortem or clear clinical evidence, we have fifty accurately noted fatal cases, and then observe that over 72 per cent. may be credited to meningitis and pyæmia taken together. But I believe that the proportion of the latter affections is still larger, and that the primary cause of these diseases, as met with in general hospital practice, might often be found in an intra-aural condition in many of those cases which are now put down as "obscure" as to origin. I feel strongly that if such cases were recognised and recorded in their true light it would be found that abscesses in the brain and cerebellum did not constitute a tithe of the usual complications of suppuration in the temporal bone. More than nine-tenths would come under the headings of meningitis, pyæmia, and sinus phlebitis. And if we included in our analysis the cases which recover the preponderance of the latter class of diseases would be still greater.

These are our great enemies, which, once they have fully de-

clared themselves, leave us very little hope of a successful combat with them. Still there is a hope, however slender, and I believe that occasionally a moderate amount of meningitis, of sinus phlebitis, and even of pyæmia, is recovered from without any elaborate operation, under careful treatment. I have seen several such cases in my own experience where special symptoms in two, at least, pointed to meningitis or sinus phlebitis, which were recovered from; and where joint affections and subcutaneous abscess left no doubt as to the pyæmic condition in others, and yet the fatal issue was staved off. Such cases serve to encourage us to hope for much more from prophylaxis in the first instance, and from operation in the second. But if operations are undertaken either for meningitis, phlebitis of the sinus, or the pyæmic condition, they must be early. Again, from a study of the cases in our list, it is abundantly evident that in many instances the cerebral or cerebellar abscess had existed for a considerable period before the fatal meningitis or pyæmia supervened, and that the latter might have easily been forestalled by operation and evacuation of the septic products.

It is by no means an easy matter, I find, in a case in which a cerebral or cerebellar abscess is associated with meningitis or phlebitis of the lateral sinus, to determine, even after death, which was the earliest condition, or whether at any time they were distinct. The primary disease in the ear can always, of course, be recognised as such, and we may satisfy ourselves that encephalic abscesses and sinus phlebitis are frequently the originating cause of meningitis, by sudden rupture or slow extension. But beyond this we are still in uncertainty. The same may be said of the relation of pyæmia to the other complications mentioned. It is quite clear that it may start from the intra-aural focus of disease, and run its course without any cranial complication. But it is also evident that it is often only the sequel either to cerebral abscess, meningitis, or phlebitis of the lateral sinus. But in any given case of pyæmia with intra-cranial complication, to determine which has been the earliest affection presents difficulties which we are not yet in a position to solve, even by post-mortem examination. What can be done clinically we shall see presently.

The age of the individuals in our list of fatal cases is interesting, and shows a perfect parallelism with the age of those suffering with the primary suppurative diseases of the middle ear. If we take any long list of the latter, observed consecutively, by far the greater number are found in patients above the age of fifteen. Bezold's yearly reports furnish us with as accurate data upon this point as could be cited. Here I find that out of 2,522 consecutive cases of otitis media purulenta seen by himself, 768 were under fifteen, and 1,754 above that age. In other words, 30 per cent. were children, and 70 per cent. practically adults. With these figures before us we might expect, *cæteris paribus*, to meet with intra-cranial complications of suppurative middle-ear disease more than twice as often among adults as among children. And when we come to examine our own list we find that the above proportion is very closely followed. Thus, out of 35 cases dying of these diseases, and two saved by opening cerebral collections of pus, at the two general hospitals receiving adults and children during twelve years, 11 were below fifteen years of age, and 26 above, showing 30 per cent. and 70 per cent. respectively. If the Ormond Street cases are added, of course the proportion is different, but this would probably not represent the true nature of the case as regards the point of age.

A further analysis of cases shows that the various diseases in question preserve practically the same proportion to one another amongst adults as amongst children.

This preponderance of serious complications among adults will probably be shown to be due to the greater development of the mastoid cells and other accessory cavities of the middle ear in full-grown persons, as well as to the larger size of the Eustachian opening in the tympanum in the child, and the consequent better drainage when pus is present in the latter in children.

The local peculiarities of the complications of ear disease which we have been considering now deserve some special attention.

Of *pyæmia* it need only be said that it may be associated with any of the intra-cranial conditions, or may start from suppuration *in the middle* ear without any mischief at all within the skull.

It is only included in our present considerations because it is so frequently a complication of one or other of the diseases in question, and affects seriously our prognosis whenever thus met with.

Phlebitis of the lateral sinus is usually limited to that part of the vessel which lies in contact with the sulcus lateralis on the inner aspect of the mastoid process (fig. 8, *c*, p. 55). But occasionally the inflammation extends downwards into the internal jugular vein, though apparently not nearly so frequently as might be expected, when we consider what occurs in other veins affected with septic inflammation. It is rare for the process to extend backwards against the current of the blood, and this only occurs, so far as we know, in those cases in which thrombosis has also taken place, then the septic process is propagated probably along the freshly formed clot, or impure blood is carried by the reflux current as the blood makes its way backwards into other sinuses.

Thrombosis, on the other hand, is not so limited, although it originates in the septic phlebitis just described. It frequently extends not only downwards well into the internal jugular vein, but also upwards as far as the torcular herophyli, or even farther into the other sinuses. Sometimes the clot formed is tough, and evidently contains but little septic material. It may solidify and organise so as completely to obliterate the vessel. In other cases it is soft and puriform at its centre, and brittle throughout, being at the same time but loosely adherent to the walls of the sinus in parts. It is obvious from this that the dangers from thrombosis, *per se*, of the lateral sinus must vary greatly; the more or less plastic thrombus must be far less dangerous than the soft aplastic clot. The latter, to begin with, is due to a far severer initial infection than the other form, and is also far more liable to break up and carry infection to distant parts. The risks of operating upon the neighbourhood of the lateral sinus where thrombosis has taken place will vary considerably with the character of the clot, as will be described somewhat more fully later on.

The situation and behaviour of *subdural abscesses* due to otitis media purulenta is a matter of great importance. In the cases which I have had an opportunity of studying two spots appear to

have been particularly liable to this condition. The first and commonest, especially among children, is the neighbourhood of the petroso-squamosal suture above. The next and most serious is the sulcus lateralis below. When pus forms under the dura mater in the first situation, as the result of the septic infection along the vessels which pass through this suture from the ear to the dura, it may spread in several directions. So far as I can make out, the most common is downwards and backwards into the sulcus lateralis. But it may also spread upwards and backwards over the inner surface of the squamous portion of the temporal bone, forming a large subdural collection. This is apparently the form of subdural abscess which has the greatest tendency to burst outwards through the squamous bone. The latter being very thin, and often softened by the same inflammation which has given rise to the abscess, is easily perforated by the pus. The point at which this rupture takes place is generally within an inch of the meatus, above and behind the latter. Here the bone gradually gives way, and the intra-cranial pus is poured out under the periosteum until an *extra*-cranial collection of large size has been formed. Such a collection has often been mistaken for an ordinary periosteal abscess, the severe symptoms accompanying it being unaccounted for until post-mortem examination has revealed the fact that it was but a secondary offshoot of an intra-cranial subdural inflammation. Again, such an abscess may burst through the roof of the tympanum in the same way, and thus drain itself effectually by the meatus. It seems probable that some of the cases in which abscesses of the brain have been stated to have discharged themselves through the ear, with recovery, have been of this nature, the brain all the time having been quite uninvolved.

The variety of subdural abscess which forms in the sulcus lateralis is due to the same process extending from the tympanum or its mastoid offshoot through the vessels of the thin layer of bone which separates the cavity of the brain from that of the ear. Its spread is usually upwards towards the surface of the petrous bone, where it may meet a similar collection. This, too, may perforate the overlying bone, or escape by the mastoid foramen behind (fig. 8, *d*, p. 55), and present exter-

nally under the periosteum over the mastoid process; but this appears to be rare. It seems more often to rupture into the lateral sinus, thus leading to fatal pyæmia. Both varieties are most frequently fatal, however, by producing a septic pachymeningitis, which ultimately leads either to general diffused inflammation of the coverings of the brain or to a cerebral or cerebellar abscess. It is in these subdural abscesses that the surgeon can be most useful, by interfering promptly with gouge or trephine, as we shall see presently.

The *meningitis* just alluded to, as starting in inflammation of the petrosal dura, appears, from clinical and post-mortem evidence combined, to be frequently limited for a considerable time to the immediate neighbourhood of the initial focus of infection. These are the cases in which we may look for good results from operation. The process appears often to limit itself by more or less of the usual plastic exudation which is observed in affections of other serous membranes. This form is usually found over the tympanum and squamous part of the temporal bone, or over the sulcus lateralis in the posterior fossa of the skull. But there is also a form of the disease which appears to have little or no tendency to limit itself. Here the process is a diffuse septic one, and analogous to what we see in the pleura and peritoneum in certain cases where the whole surface is quickly invaded, without the production of any plastic lymph. The difference between the two kinds of inflammation is probably due to the particular form of organism which initiates the septic process in the ear, just as in other parts of the body analogous differences in plastic and aplastic inflammations may be observed. The aplastic form has a greater tendency to spread over the base of the brain than over the vertex, as might be expected, considering the part which gravity plays in distributing septic fluids. It has also, in several of the cases in our list, extended into the spinal canal even as far down as the cauda equina.

As to the position in which *cerebral abscesses* are usually found as the result of ear disease, very little need be said. They are almost invariably seated in the temporal lobe, behind a line drawn vertically through the tragus (fig. 1, p. 36). From this they may extend back-

wards into the occipital lobe to a greater or less extent, and very rarely forwards towards the apex of the temporal lobe. But these exceptions are so uncommon that they need hardly be considered for practical purposes. The vertical line just mentioned, and

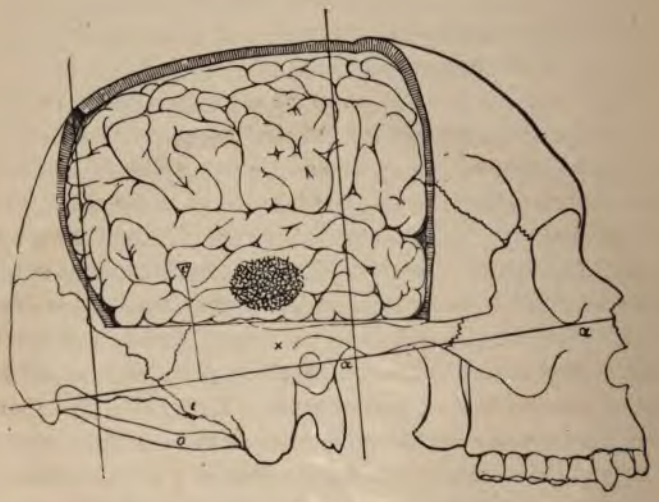

Fig. 1.

(Kindly lent by the publishers of the author's "Manual of Surgical Operations.")

From a photograph of a Skull laid open, to show the situation of Temporo-sphenoidal Abscesses (the shaded spot). *a a*, "Base Line." The two vertical lines drawn at right angles to the latter will include all such abscesses. ×, the spot for Trephining in opening the Mastoid Antrum.

another two inches behind it and parallel to it (fig. 1), would include the greater part of nine-tenths of these abscesses. As I have said elsewhere, a circle with a $1\frac{1}{4}$ inch radius, having its centre an inch above and behind the meatus, would include the greater part of most of these, and from within this circle they would all be reached by trephine and trochar.

Here, again, we meet with the same differences as to limitation

of the septic process which have just been alluded to in referring to the other diseases of our group. We have the well-defined abscess, limited by a lining membrane, and the diffused inflammatory softening, with no trace of bounding lymph. From want of sufficient accuracy in detail in the records before me, it is not possible to say what the relative frequency of these two forms is,

Fig. 2.

To show the position of Cerebellar Abscesses in the anterior part of the Lateral Lobes (pale spot). A vertical section of the Encephalon has been made to expose the space over the Island of Reil and its convolutions, 1, 2, 3, 4. (After Dalton.)

however desirable it might be that we should know. For it seems obvious that the well-defined abscess ought to be much more within the reach of operation than the diffused softening.

The contents of these abscesses also vary much, being in some cases made up of thick greenish and intensely fœtid pus, and in others of thin pale inodorous fluid hardly purulent. I think it not

improbable that those containing thick fœtid pus will prove more amenable to treatment than the others.

In the case of *cerebellar abscess* we find the same variety, too, as to limitation and diffusion of the inflammation as in the case of the temporal lobe, and presumably for the same reasons. The habitat of these abscesses is, fortunately for the operator, a very constant one. Cerebellar abscesses, due to ear disease, are almost invariably seated in the anterior portion of the lateral lobes, where these lie in contact with the posterior surface of the petrous bone and lateral sinus (fig. 2, p. 37). Some of these tunnel backwards in the upper part of the lobe in a peculiar manner, but most of them lie about at the mid-level from above downwards. It is rare to meet with cerebellar abscess without very extensive mischief either in or under the dura mater of the sulcus lateralis or about the internal auditory meatus. These abscesses, like those of the temporal lobe, have been known to work their way to the surface through the overlying inner wall of the mastoid cells, but I cannot find a case in which recovery has been brought about in this way.

We must now endeavour to find out whether these various complications of suppuration in the ear can be distinguished from one another by their clinical symptoms. In making this inquiry the list from the three hospitals has furnished us with some of the most accurately recorded cases to be found. But I have not only drawn upon these, but upon a far larger mass of cases collected wherever they have been recorded with any approach to scientific accuracy. In reading these carefully, and in studying besides a considerable number at the bed-side, both in hospital and in private, I must, of course, have gathered certain impressions which it is impossible at present to reduce to cut and dried deductions, but which nevertheless would certainly sway me in dealing with such cases clinically. These impressions must influence what follows here and there, and may, I trust, be of some value as coming from one who has approached the whole subject without any special bias.

As already noted, these dangerous sequelæ are rarely the consequence of a *primary acute* attack of inflammation in the *middle ear*. The patients have almost invariably had the

otitis media for a considerable time. They may be in excellent general health, and suffer not in the least from the slight discharge from the ear. Then suddenly the latter feels hot and uncomfortable, and very frequently *ceases* to discharge. If the fundus of the meatus be now examined it is just as common to find no cause of obstruction as to detect a polyp or swelling of the meatus damming back the discharge. Where there is no obstructing material the cessation of the discharge is probably analogous to that which is seen in wounds when attacked by some of the septic infections, such as erysipelas. The general symptom of malaise is then observed, sometimes with, and sometimes without a rigor. At this stage the temperature is sure to be raised, no matter what is to follow. The patient may also vomit, or feel nausea at least. Acute pain in the ear may or may not be complained of, but dull aching is usually present in and about the mastoid region, and often down the side of the neck, and radiating up the temporal region. Aching in the back and limbs is also not uncommon. The pulse will be frequent, the tongue often foul, and at an early stage there is often an attack of diarrhœa.

Everything in these symptoms, in short, points to an acute septic infection grafted on an ordinary saprogenic suppuration. These symptoms do not always, of course, run on to one or other of the complications we have in view, but when they do so we generally observe severe localised headache supervening upon them. This headache is, as a rule, dull and heavy or "splitting." As to its situation, the greatest variety has been noticed even with the same intra-cranial lesion. Thus, cerebellar abscesses have frequently been associated with frontal headache, while temporo-sphenoidal abscesses have produced occipital pain; in meningitis there has sometimes been no headache at all, and at other times this has been severe all over the head, with an entirely localised process. Closer observation of this symptom, and accurate record in the future, may possibly enable us to form more definite rules for guidance. But so far as we know at present, we must be very cautious in drawing conclusions from the situation of the pain complained of. I think, however, that this much may be

said, that the pain in meningitis is usually much sharper and more general, while that of abscess is duller and more local.

Tenderness, too, may be very deceptive. Thus, in my own second case, where a collection of inflammatory fluid was evacuated from the middle of the Sylvian fissure over the island of Reil (fig. 3, 2 and 3), with recovery, there was most acute tenderness all over the affected side of the head, no one spot being more definitely painful than another. Of course when there is well-marked subdural abscess we may expect to find

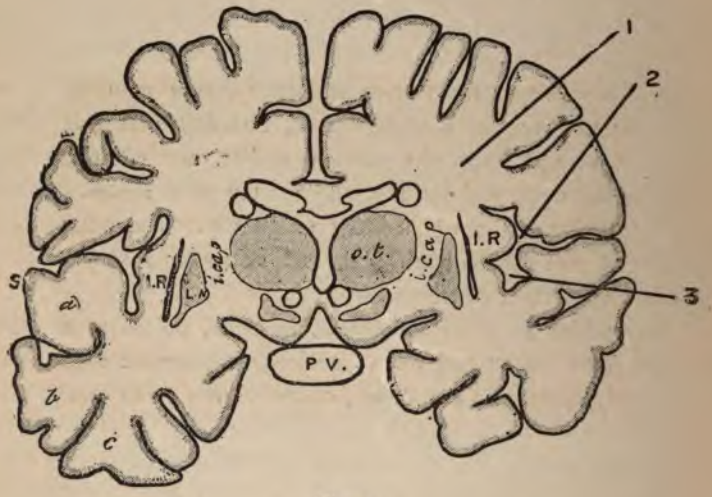

Fig. 3.

Vertical Transverse Section of the Brain, to show the Space over the Island of Reil (I.R.) which was tapped and drained in my second case. The various Convolutions and Conducting Tracts are indicated by letters. (After Dalton.)

a considerable amount of tenderness and swelling over the post mastoid or temporal region. Or if there be a cerebral abscess, pure and simple, careful percussion of the skull will probably elicit greater sensitiveness over the affected area than elsewhere; and this will be valuable evidence if taken with the other symptoms. I fear that beyond this the records hitherto pub-

lished furnish us with but few reliable data. Still, in the future we may hope much may be brought out by more careful observation.

But the character of the patient's temperature affords very valuable evidence as to the nature of the complication in any particular instance. This comes out clearly in examining our series of cases, and has long been recognised. The chief source of difficulty in this observation lies in the fact that pure types of the diseases we are considering are rare; they are usually met with

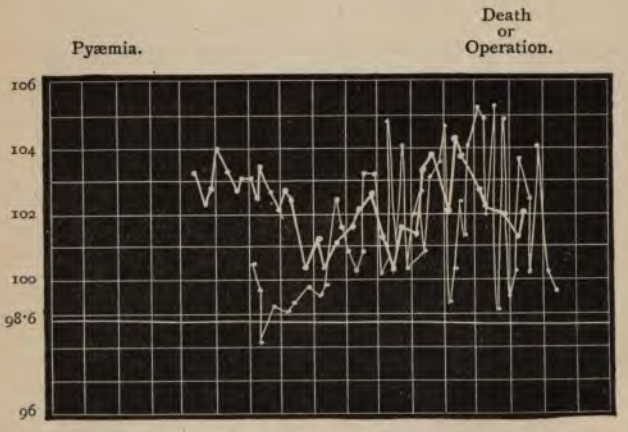

Fig. 4.

Pyæmic Chart, including the records of several cases. The temperature lines are of extreme irregularity and the oscillations frequent. Each space represents one day in all the charts.

in combination more or less complex. But taking the simplest cases we can find of each disease, we recognise important differences in their temperature charts. That of *pyæmia*, for instance, soon leaves us in little doubt (fig. 4). The oscillations between subnormal and the highest ranges, occurring without any relation to the time of day or the escape or retention of discharge, are as characteristic here as when the focus of infection is in any other part of the body. Such a chart, if accompanied by recurring

rigors (usually noticed on the ascending curve), can hardly be mistaken.

The nearest approach to the pyæmic temperature is that seen in cases in which *phlebitis of the lateral sinus exists.* Of course this affection is one of those most commonly associated with pyæmia. But there is no doubt that it may exist for a considerable time without the latter, and ultimately be recovered from. In such cases the extremes reached by the oscillations of temperature are not so great; the common initial rigor is not repeated more than once or twice, unless the case run on to pyæmia, and

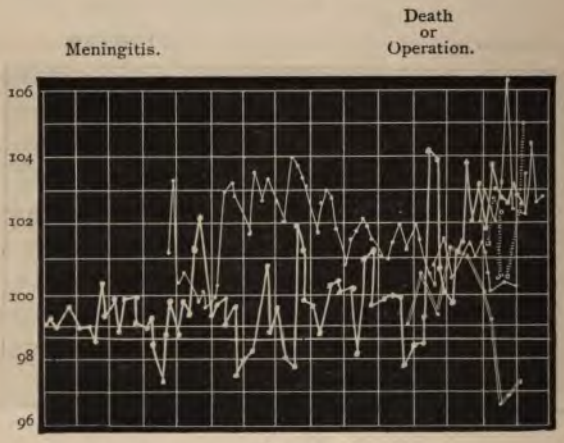

Fig. 5.

Meningitis Chart, including several cases. The temperature lines are not so irregular as in pyæmia, but often higher.

ultimately the temperature curve becomes more steady, until it subsides to normal.

In *meningitis*, although the temperature is very high, the chart (fig. 5) shows a considerable contrast to that in pyæmia, in the absence of that extreme irregularity so well known to us all. In a simple case, then, of general meningitis the temperature will be persistently high, as a rule; but in a localised process resembling more the formation of an abscess on the surface of the brain I should *expect it not to* be high, and to be steady.

As to the chart of *subdural abscess*, I do not think that we have any evidence of peculiarity. The fever is high and steady if the case be uncomplicated, which is not common.

It should not be forgotten that simple inflammatory disease of the middle ear, without any intra-cranial complication, may produce febrile disturbances with all those shades of difference just alluded to, and to exclude this cause in any given case is not by any means an easy matter without operation.

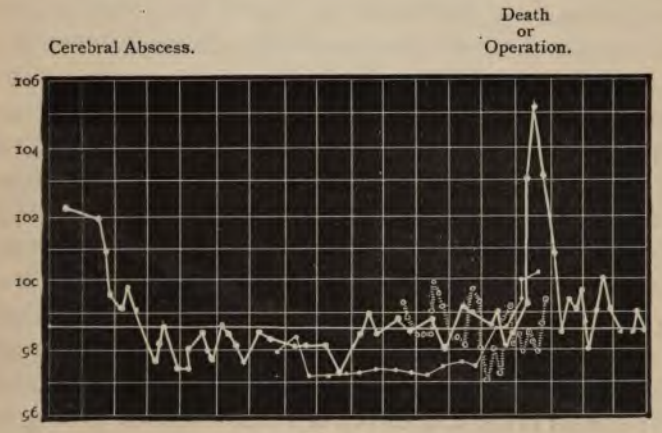

Fig. 6.

Temperature Chart of Cerebral Abscess, including several cases. The temperature lines are unusually low, often subnormal, and do not show much oscillation.

When we turn to the temperature chart of *cerebral abscess* we notice something very characteristic (fig. 6). After an initial rise there is usually a steady fall until the abscess is well formed, and then the daily records are about or below the normal. This is a remarkable fact, and one which has long attracted attention, having first, I believe, been emphasised by Mr. Hulke. It is the more startling when we remember that the process which originates the low temperature is primarily an inflammatory one, and that in proportion to its full development is the reduction of body heat.

The latter only rises to the normal standard when the abscess is evacuated. This was well seen in the first of my own cases. As a rule, however, where there is a cerebral abscess some other complication, such as meningitis, coexists with it at the time when the case first attracts serious attention, and thus the typical chart is not produced. But where the only lesion is an abscess the type mentioned is well seen as in fig. 6.

The same rule as to temperature probably holds good for *cerebellar abscess;* but here it is much rarer to find the lesion a simple one; there is in the majority of cases a localised meningitis besides, or a phlebitis of the lateral sinus, and thus our opportunities of generalising from simple inflammatory lesions of the cerebellum alone are very limited.

Of the state of the *pulse* in these various lesions no cut and dried description can be given. We know, however, that as a rule the simple cerebral abscess is accompanied by a slow pulse of even rhythm and good volume; also that meningitis produces a rapid pulse of small volume, and often considerable irregularity, which increases as the case approaches a fatal termination. But as to the other complications of ear disease, I cannot say that the study of all the recorded cases within reach has enabled me to recognise any characteristic differences between the pulse of one and another.

The *bowels* in the case of cerebral and cerebellar abscess and in meningitis are, except as death approaches, usually confined, often obstinately so, but in the other diseases of our group where the element of septic infection is well developed there is usually no constipation, and there is often diarrhœa. As regards the value of this symptom it may, I think, be generally laid down that where the bowels are regular, or if diarrhœa is present, simple meningitis and isolated encephalic abscess are excluded. If these lesions exist in such a case they are probably overpowered by some of the other complications in an advanced condition, which include septic absorption, the diarrhœa being an eliminatory effort. Again, if persistent constipation be a prominent symptom it may be held to point to either meningitis or encephalic abscess as a dominant lesion. Beyond this we learn *little that is* definite from the cases included in our survey.

In considering the diagnostic value of the symptom of *vomiting* I venture to think that hitherto sufficient care has not been taken to distinguish between the sickness incidental to the primary septic infection and that due to the more local effect of a collection of inflammatory matter within the encephalon. I have carefully studied our series of cases with special reference to this point, and have found that severe sickness is common to each and all of the complications of temporal bone disease at the onset of the attack. And it is just as severe in pyæmia or phlebitis of the lateral sinus where there is no intra-cranial collection of pus as it is even with cerebellar abscess. And when we admit the justice of the common belief that vomiting is more frequent with cerebellar abscess than with temporo-sphenoidal abscess we must not forget that this is *possibly* due to the more common association of phlebitis of the lateral sinus, and consequent septic absorption, with the former condition than with the latter. Septic infection of any part of the body is commonly, we know, ushered in by nausea and vomiting, which often persist in severe cases. With inflammation, then, in the neighbourhood of one of the largest veins in the system, which the lateral sinus is, abundant septic absorption is just what we should expect, and vomiting is consequently common whether there be any abscess or not. In any given case, therefore, in estimating the weight to be given to this symptom, we must try to distinguish between the primary effects of the inception of the poison into the circulation and the local effects of an abscess, whether these be due to pressure or irritation. The most reliable help in this matter is the temperature. If the vomiting appear early in the attack, and be associated with much fever, it is more likely to be due to one of the affections of the lateral sinus with septic infection than to encephalic abscess. If, on the other hand, it appear later on, with a normal or sub-normal temperature, the vomiting is more likely to be due to abscess in the brain or cerebellum. In meningitis, however, we often have early vomiting, with its usual high temperature; but in this case the rapid onset of delirium or coma will probably help us to decide against pyæmia at all events, especially if there are no recurring rigors.

The value of *rigors* alone as a symptom is not great. They occur, as we know, in a great variety of inflammatory affections in other parts of the body, and so it is here. We are particularly familiar with them in pyæmia from temporal bone disease, when they usually recur irregularly. But they are also often observed at the onset of an attack of sinus phlebitis, of subdural abscess, and of encephalic abscess. It is only when their presence is carefully considered in conjunction with other symptoms that we can form any estimate of their bearing.

Delirium, too, is evidence of comparatively little value in differential diagnosis. It is observed in nearly all the diseases of this group in their last stages. But this much may be said, that it is more likely to be observed early in meningitis than in any of the rest of the group. In abscess of the brain and cerebellum it is often absent, coma being more common, while in septicæmia and pyæmia the sensorium is frequently quite clear until immediately before the end of life.

Slow and sluggish cerebration, on the other hand, should always be looked out for, as it rather indicates an encephalic abscess than any other condition. I believe this is sometimes mistaken for deafness in such cases. The patient, when spoken to, appears not to have heard or comprehended for a few moments, and then commences to reply very slowly and deliberately, but quite correctly. This was a well-marked symptom in one of my own cases. The patient, who was naturally an intelligent man, on being asked a question, looked straight at the interrogator as though to take in what was said, and then, after a pause, answered slowly, but quite correctly. It is a point of some interest in connection with this case that the curator of our museum at University College, where this patient has been a porter for several years, remarked to me that the evacuation of the abscess from his brain appeared to have immensely benefited his intelligence; that whereas for a considerable time before the attack for which I operated the patient had been stupid and lethargic about his work, he has ever since been particularly intelligent and energetic. It is quite possible, and indeed very probable, that *this man* suffered from his temporo-sphenoidal abscess for a con-

siderable time before he was operated on, and that this lethargy and slow cerebration were some of the results of it. Whether this effect be due to simple intra-cranial pressure, or to the presence of the decomposition products of the abscess among the nervous structures, remains for future experiment to decide. But whatever the cause, the recovery of the mental powers is remarkably rapid and complete after evacuation of the pent up matter. It is worth noting that this symptom is present just as markedly with cerebellar abscess as with collections in the temporal lobe, as we learn from a detailed examination of the cases in our list; also that it is not a symptom of the other complications of temporal bone disease.

All the evidence in my own first case pointed to the conclusion that the lesion was simply an abscess in the temporal lobe without any further complication. The sluggish cerebration was present for some time before the operation, but gradually deepened into coma just before the latter was resorted to. A day or two later, when the abscess had been emptied, it was quite gone, and the contrast of the patient's condition mentally was very striking. This state is probably due to the same causes which operate in producing coma, whatever these may be, and is, I think, to be considered simply as an early stage of the latter. Taken together with other evidence, it will, I think, be found an important symptom.

The consideration of some other clinical features of these diseases must be deferred, I see, until my next lecture.

LECTURE III.

RESUMING our examination of the clinical features of the diseases under consideration, the symptom of *emaciation* must next be noticed.

Great and rapid wasting of the body has long been known as particularly frequent in some affections of the brain; and where there is no high fever or diarrhœa to account otherwise for it, it must be regarded as a symptom of considerable value. But we have seen that in typical uncomplicated cases of abscess of the brain the temperature is usually low. In a suspected case, then, with constipated bowels, slow cerebration, headache, and low temperature, especially if the latter be subnormal, our suspicion of cerebral abscess as the complication of the ear affection is strengthened by the appearance of rapid emaciation. In the absence of fever, diarrhœa, and vomiting it is not easy to account for this loss of tissue. We can only conclude that the assimilative powers are almost or entirely in abeyance, owing to the cerebral lesion, and that probably the loss of moisture from the surfaces of the body does the rest. The intense fœtor of the breath noticed in most of these cases probably indicates a complete suspension of digestion, with decomposition of any food remaining in the stomach, and if this be so, rapid emaciation is not hard to understand.

Of course with the high fever of meningitis, pyæmia, or the other septic complications, many of which will probably be associated with diarrhœa and vomiting, wasting has no special significance as a diagnostic aid.

Together with the great loss of flesh noticed with cerebral abscess, there is often associated a peculiar loss of colour or "muddiness" of the skin. This may assist our diagnosis somewhat *if present* in cases not pyæmic, for it does not appear to be

a marked symptom in any of the lesions of our group except cerebral and cerebellar abscess and pyæmia, and the temperature of the latter distinguishes it from the other two conditions.

Of the *respiration* much need not be said. In uncomplicated cases of abscess, whether of the temporal lobe or cerebellum, it is almost invariably slow, shallow, and regular. Where there has been, in addition, much basal meningitis, especially if of the posterior fossa, the respiration, besides being slow, has, in several cases, been noted as irregular, and of the Cheyne-Stokes type. In ordinary meningitis of the convex surface of the brain breathing is usually much increased in frequency and irregular; and with the other complications, too, increased frequency is the rule, though the rhythm is not necessarily disturbed.

Of *optic neuritis* the evidence in hand only justifies us in saying that where it is present and increasing it strengthens the diagnosis of intra-cranial inflammation. But as to the variety of the latter it gives us no clue. We now know that it has been present in a marked degree in cases of cerebral abscess, of meningitis, and also of thrombosis of the lateral sinus due to phlebitis where, after death, the brain was found to be quite healthy. But it has also been entirely absent in typical cases of the same diseases. I have, moreover, seen it in the most intense form in a case of subdural abscess of the sulcus lateralis, which I opened and drained with a perfectly successful result. In this case there was no reason to think that any other lesion of the structures within the skull existed, and for several weeks after the boy was well and had left hospital the condition was watched by myself and some of my colleagues, as a particularly striking instance of the affection.

We must therefore be cautious about laying too much stress upon the value of optic neuritis in differential diagnosis, until we have watched a much larger number of the diseases of our group from their commencement to death, and observed more closely the conditions under which this ocular symptom is present or absent.

With regard to changes in the *state of the pupil* in the various intra-cranial conditions under consideration, I have carefully tabulated the notes referring to this symptom in 24 cases in which a record appears. So far as this table goes, it is quite clear that no

special rule as to the lesion can be formulated from the size or reactions of the pupil. In cases apparently of exactly the same disease the condition of the iris has shown great variety, and in totally different intra-cranial conditions has been found to present either no changes at all or similar deviations from the normal. This is far from saying that it is not worth while to observe the size and reactions of the pupil in every case, because I believe that evidence as to cerebral irritation, pressure, etc., may be forthcoming in particular instances which may prove of use taken with other symptoms. But I repeat that, to judge from the records of our series of cases, no guiding rule can be formulated as to what change in the iris is to be expected with this or that lesion. Possibly closer attention to this subject in the future may yield better results.

Of the immense value as a symptom of *convulsive movements* of sets of muscles or of limbs it is hardly necessary to speak a length. If present, there is no symptom of greater value in the localisation of the lesion. We must not, however, forget that inflammatory destructive changes of both brain and cerebellum may be unaccompanied by any spasms at all. The ordinary temporo-sphenoidal abscess is the best illustration of this. Here not only are spasms absent, as a rule, but it is in many cases impossible to detect any deviation from the normal nervous phenomena of any part of the body. In my own first case, in which Dr. Gowers asked me to operate for temporal abscess, there had been, I need hardly say, a most exhaustive examination as to the condition of the patient's nervous system. But neither motor nor sensory disturbances of any kind were detected, nor were the reflexes in any way abnormal. Indeed, except for an unprovoked attack of vomiting, and for double optic neuritis and slight inequality of the pupils, nothing specially traceable to nervous disturbance was observed at all.

Next to local spasm, *paralysis* of motion and sensation is, of course, a most valuable symptom. And if limited, and following upon a previous localised convulsion, as is often the case, it is evidence both as to the character and situation of the intra-cranial lesion such as cannot be surpassed in value. In my own second

case of operation for an inflammatory collection in the brain the seat, nature, and spread of the disease were clearly mapped out by the initial epileptiform fit, then paresis on the left side of the face, later twitchings in the left wrist, followed by partial paralysis of the arm, and finally by loss of movement in the left leg. Here I believed the inflammation to have led to a collection of meningitic

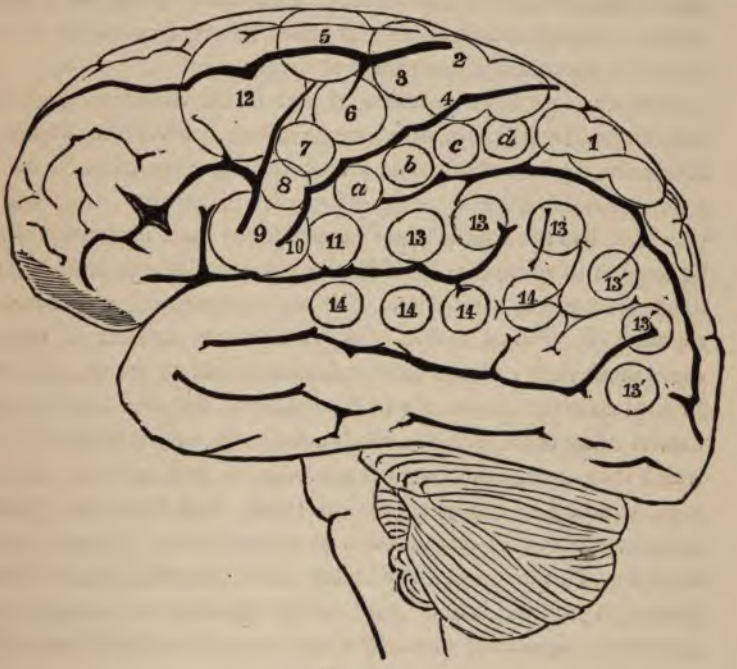

Fig. 7.
(After Ferrier.—Kindly lent by Messrs. Smith, Elder, and Co.)

fluid about the middle of the Sylvian fissure, and to be spreading up along the fissure of Rolando, involving in succession the face, arm, and leg centres (fig. 7, 6 7, *a b c d*, 1 and 2). And operating upon this hypothesis, the collection was reached and evacuated with a perfectly successful result, the patient being present here to-day.

Of the value of changes in the condition of the superficial and deep *reflexes* I cannot speak with any authority. I can only say that in the limited number of cases of these particular lesions which have been under my notice the state of the reflexes has afforded very little evidence of practical value, even when interpreted by those possessing a special familiarity with these nervous conditions. And from a study of the cases in our list, it would appear that this has been a common experience so far as I can gather. I speak, however, on this point subject to correction; it is one for specialists in neurology to determine.

Now, when we come to review as a whole the various symptoms, objective and subjective, which we have just been considering individually, we cannot fail to be struck with the very limited assistance obtainable from them for differential diagnosis. And those who have had the largest experience of these cases in practice will be the first to admit that the difficulties surrounding the differential diagnosis are here very great. For my own part, although I have watched the clinical course of a considerable number of these cases in all their varieties closely, I believe that we are more likely to form an accurate estimate of the nature of the affections from a careful study of the primary conditions in the temporal bone from which they spring, and from a knowledge of their general pathology, as noted in the post-mortem room, than from any *special* symptoms which we may observe at the bed-side. Moreover, do what we will, the uncertainty in any given case will always be so great that nothing but an exploratory operation will clear it up. How such exploratory operations may be conducted with least risk to the patient will be indicated presently.

Turning now to the *treatment* of the diseases in question, it hardly seems necessary to insist upon the pressing need of more general attention to a stringent prophylaxis. And yet from all that one sees daily, it is here that medicine and surgery appear weakest in practice. If it be true that suppurative ear disease is common and that if neglected at least two or three out of every hundred persons affected with it die, sooner or later, of various complications; and if it be true that under well-directed antiseptic treatment suppurative otitis media can be arrested, it is obviously the duty

of every medical man meeting with such disease *to attack it in every case at its outset vigorously, and not to relax in his efforts until the inflammation has entirely and definitely ceased.*

Cases will, however, present themselves to us from time to time where no such treatment has been carried out at all, or has been ineffectual, and one or more of the intra-cranial complications described have developed. In such a case it is, I fear, vain to hope for anything from the administration of drugs or local applications. Nothing short of operative interference holds out any prospect of safety for the patient. As long as septic matter is pent up either in the temporal bone or inside the skull it is waste of time to give drugs or apply leeches, heat, or cold locally. The only line of treatment which affords a hope of success in these very serious cases is to gain access to the focus of infection in the middle ear in the first place, and to thoroughly cleanse it, and in the next to search for the secondary inflamed area within the skull, and cleanse and drain it likewise. Antiseptic irrigation and fomentation are of the utmost value once the foci of inflammation have been reached by removal of bone, but before this has been done must obviously be quite ineffectual whether the pus lie in the mastoid cells, the sulcus lateralis, or the brain.

But it may be urged that for some of the complications of ear disease no operation can hold out a prospect of relief. Of course this is true of the *last* stages of *all* those affections enumerated. But the operative achievements of recent years have amply demonstrated that even with advanced intra-cranial disease of the kind now before us recovery is not impossible, if only a free escape of the septic matter is secured by operation on the bony case which confines it.

From the records of recent surgery we can now prove that operation has saved the lives of many patients affected with one or other of all the common intra-cranial complications of disease of the temporal bone except cerebellar abscess. There seems also to be every reason to hope that in the near future the latter condition will prove as amenable to operative interference as any of the others.

Since these lines were written I have received a letter from

Glasgow, announcing that Dr. Macewen had opened the cerebellum within the last couple of weeks, and evacuated about four ounces of pus. The patient, who was apparently dying at the time, is now convalescent.

But even my own experience of opening the skull for these lesions, which is not yet large, embraces operations for cerebral abscess with recovery, for localised meningitis with recovery, for subdural abscess with the same result, also for the pyæmic condition with swellings over the joints, and subcutaneous abscesses with recovery. I cannot claim to have operated successfully for cerebellar abscess, but I believe I have succeeded in arresting the course of an attack of phlebitis of the lateral sinus in one or two cases, as others have in numerous instances.

We are justified, therefore, in indulging in far greater hopefulness for the future of these diseases than we have felt in the past, especially if they are recognised earlier than heretofore, and if vigorous and well-directed action is taken to give free vent to all retained inflammatory products, no matter where they may be, the most aggressive antisepsis being maintained throughout the whole course of the treatment.

I shall now endeavour to sketch, briefly, the line of action which may be recommended to the operator for each of the several conditions under special consideration. And in expressing my own opinion upon these points it is not without due regard to what has been said and done by others, both at home and abroad, with whose work I have spared no pains to make myself familiar.

Taking them in the order in which the disease is likely to spread, we shall first consider the treatment suitable for a case of *phlebitis of the lateral sinus* in a fairly early stage—that is, before a hopeless state of general embolic pyæmia has become established.

In the first place, it goes without saying that the whole of the middle ear, the primary seat of the septic inflammation, must be thoroughly cleansed and drained. Now, to do this completely through the external meatus is impossible in those cases in which the disease has extended backwards towards the sulcus lateralis, *and has invaded* the mastoid bone on its way. It is necessary,

then, to make a free opening through the latter into the middle ear, so as to have the whole area of primary infection under the control of irrigation. This opening is best made into the mastoid antrum, at a point (fig. 1, ×), (fig. 8, *x*) from a third to half an inch behind, and the same distance above the centre of the meatus of the

Fig. 8.

(Kindly lent by Messrs. Longman, from the author's "Manual of Surgical Operations.")

Temporal bone, with section to expose the mastoid antrum, *x*, and the cells below it. *c* shows where the sulcus lateralis has been laid bare. *a* represents the "base line," *b* the centre of the meatus, *d* the foramen for the mastoid vein opening into the sulcus lateralis.

ear (fig. 8, *b*), *i.e.*, above the "base line" (fig. 8, *a*). The incision in the soft parts to be preferred for this operation is a vertical one behind the insertion of the auricle, which may be met, if necessary, at its middle by another horizontal cut at right

angles to it from behind. The two triangular flaps thus formed being turned back, the external wall of the antrum mastoideum is completely gouged away downwards and forwards until the cavity is laid freely open. Through this aperture the whole of the middle ear is thoroughly washed out with some strong antiseptic fluid, until the latter runs out clear through the external meatus. When thoroughly clean I find it well to inject the whole cavity with iodoform emulsion, which deposits the powder over all the affected surfaces. Boracic fomentations are then applied and frequently changed. If by these measures the symptoms are not speedily relieved the mastoid process should be freely gouged away until the membranous lateral sinus is well exposed. With care in removing the bone layer by layer, no damage to the vessel is done. But even if wounded the result is not necessarily fatal, as many recorded cases prove, provided only the injured spot be at once covered with a pad of antiseptic fabric, dusted with iodoform. The dura mater, containing the sinus, may in this way be exposed over a considerable area with great advantage. When this has been done all pent up matter will be evacuated, and the removal of the inflamed bone will drain the area of infection. Any septic products, too, left behind, will have a tendency to escape into the loose gauze dressing or hot fomentations now applied. The exciting causes of the phlebitis will therefore be removed, and as is shown by many recorded cases, the prospect of recovery is thereby greatly enhanced. Recovery has followed these operations, even in cases in which repeated rigors gave good reason to believe that general pyæmic infection had commenced; so that rigors alone need not contra-indicate the measure, but (except in the last stages of exhaustion) rather stimulate us to surgical interference.

I cannot help thinking, sir, that we have been inclined hitherto altogether to overrate the risks of operations in the neighbourhood of the lateral sinus, and that much has been lost thereby. Before the introduction of the antiseptic methods into general use it may have been natural to regard this region with peculiar dread. But now that the dangers of infecting our own wound have been eliminated the question of danger almost entirely

depends upon the manipulative skill of the operator. It ought to be no more difficult to remove the bone layer by layer, so as to expose the dura mater of the lateral sinus, than it is to expose the dura mater in any other region of the skull. We cannot, of course, trephine here with the same precision, for we cannot by any means ascertain the thickness of the bone. Moreover, the inner surface is too irregular in shape for the safe use of the trephine, which might easily penetrate the vein at one spot, while the bone at another was hardly cut. But with gouge or chisel there is nothing to prevent our gradually removing enough bone in layers to expose the sinus without any injury to it. And once it is visible at one spot, the operator ought to be able to expose it freely without further risk. As a matter of fact, it has been laid bare over a large area in several cases with the greatest benefit to the patient, the whole of the mastoid portion of the temporal bone having been completely removed.

If the bone is freely excised it is better, perhaps, to leave the flaps unstitched, in which case no drain-tube is required; but where only a limited opening has been made in the bone I personally prefer a silver or leaden drain-tube, which can be much more easily introduced and kept in place than a rubber one.

For *thrombosis of the lateral sinus* caused in the way already referred to, and for *subdural abscess*, the line of action is practically the same as in the last case. But remembering that the pus is very likely to be found in greatest amount over the squamoso-petrosal suture above, or inside of the mastoid process in the sulcus lateralis behind, our removal of bone must be freer in both these directions until the dura mater is well exposed, and all question of the release of septic matter beneath it is set at rest. We are far more likely to err in the direction of not removing enough bone than in removing too much. Except the lateral sinus, there are no structures of any great importance in the neighbourhood likely to be injured, and the removal of a little more or less bone is a matter of no consequence. In one case in which I operated successfully for subdural abscess in the sulcus lateralis the diagnosis was much facilitated by observing that on exposing the mastoid region of the skull pus welled out of the fora-

men for the mastoid vein (fig. 8, *d*, p. 55). This fluid must have been pent up in the sulcus lateralis, and have been released as soon as the bone was stripped of its soft parts. It was only necessary in this case to enlarge the foramen slightly to evacuate all the septic products completely. But from dissections in fatal cases it is quite clear that there might be a considerable amount of pent up matter under the dura mater, too thick to flow away through such a small opening, and in such a case free removal of bone will always be necessary. Indeed, I think that this ought to be the rule whenever a subdural abscess is discovered. In many cases the material found here has been curdy, inspissated, fœtid pus, which nothing short of instrumental removal, together with free irrigation, could have dislodged, and which without operation would never have worked its way to the surface.

It will probably occur to many that where there is a septic phlebitis of the lateral or petrosal sinuses any operation in the neighbourhood will give rise to increased risk of some portion of infected clot being dislodged and carried into the circulation, setting up embolic changes in heart or lungs. Undoubtedly there is this risk, but against it must be set off the hopeless nature of the condition if left unrelieved. Moreover, in this particular instance we are operating upon rigid bone tissue, and the soft fibrous covering of the sinus is not necessarily squeezed or dragged during the removal of the bony covering, as it might be if we were operating for the exposure, say, of an inflamed or thrombosed internal jugular vein. There ought not, therefore, to be as great likelihood of portions of adherent septic lymph being broken off the sinus wall as there would be in the latter case. Great care, however, is obviously incumbent on us in gouging away the bone in order to open up the sulcus lateralis, not to encroach upon the membranous sinus, or in any way to disturb it. Our aim should be simply to lay bare and drain thoroughly the subdural collection of pus around it, and experience proves that this is possible and will save life.

We turn now to the question whether *meningitis* is amenable to operative treatment. My own opinion is that up to a certain *point, while* the process is still fairly localised, surgical inter-

ference may save patients otherwise inevitably doomed. Everything depends, of course, upon the direction in which the process is spreading, and whether it is of the more diffuse and septic form or not. For there is ample post-mortem evidence before us to prove, as I have already stated, that there is as much variety in the forms of inflammation of the arachnoid as of the other serous membranes of the body, and that we may meet with meningitis in one case having a strong tendency to localise itself over a limited area in spite of abundant exudation, while in another the process is essentially different, having a tendency rapidly to diffuse itself, and that with a minimum of lymph production. This point seems to be overlooked sometimes in the consideration of the feasibility of arresting inflammatory processes within the skull. Now, in the case of the diffuse infective meningitis it would be as irrational to expect to arrest the disease by operation on one spot of the infected area as it would be to arrest the spread of a cutaneous erysipelas by excising a portion of the inflamed skin. But in the other cases of localised meningitis, with more or less plastic limitation, although the process is distinctly septic, we may expect in the future to do a good deal. Here the same general rules guide us as in a case, for instance, of phlegmonous cellular erysipelas, where, as we know, incision and evacuation of the pent up septic matter are often followed by cure if done early enough.

If the meningitis is spreading over the base of the brain towards the great centres, and where we cannot follow it, little or no benefit can result from operation. But if, as is often the case, the disease spreads from the roof of the tympanum up over the temporal lobe into the Sylvian fissure, I cannot but think that free removal of bone with antiseptic irrigation and drainage would save many a patient otherwise in a hopeless condition. In one of my own cases, in which I diagnosed this form of disease and operated for it, the result exceeded all expectations. The difficulty here will always be the diagnosis; but in view of the gravity of the prognosis, if the case is left to run its course an exploratory operation at least, which will clear up the nature of the disease, appears not only justifiable, but called for.

In those cases, therefore, in which we are led to expect meningitis, but where no very definite localising symptoms are present, I would venture to recommend the use of the trephine over the lower and anterior part of what may be called the "dangerous area." This I have defined elsewhere to lie within a circle with a $1\frac{1}{4}$ inch radius, having its centre $1\frac{1}{4}$ inch behind and above the middle of the external meatus of the ear (fig. 1, *c*, p. 36). When in this spot the dura mater has been cautiously exposed and divided the state of the serous surfaces will determine us whether to go farther or close the wound at once. If meningitis of the more plastic form be present it may be necessary to remove several circles of bone, in order to cleanse and drain the surfaces thoroughly; and remembering the history of trephining, we need not hesitate to do this largely. But in those instances in which very definite localising symptoms are present, as in one of my own cases, the spot at which the disease is affecting the brain most deeply is the spot, of course, to aim at. From the opening made here in the skull we may learn the state of the brain elsewhere by inspection, or, if necessary, by puncture, and should be prepared to remove as many circles of bone as may be necessary thoroughly to cleanse the affected area. If the openings be made near one another, but not continuous, a considerable surface of the brain may be exposed and cleansed by irrigation, without depriving it entirely of its support, which is kept up by the intervening bridges of bone. After irrigation and dusting with a light cloud of iodoform, the wounds may be partially closed, room being left for the free escape of discharge. Then a loose moist dressing of sal alembroth gauze covered with oil silk is applied, and well covered in its turn by loose packing of the same material dry. The patient should then be placed with the head lying on the affected side, to encourage the free escape of septic matter as far as possible.

That the brain is more tolerant of meningitis than is commonly supposed will, I think, be clear to any one who will examine any large mass of pathological evidence bearing upon the question. Studying this we must be struck with the large amount of disease *which has in many cases* been necessary to destroy life. Even

where the base of the brain has been involved the morbid process has often advanced to an extreme extent before the fatal result has been brought about. Is it not fair, then, to suppose that a moderate amount of meningitis, especially when spread over the lateral aspects of the encephalon, may be tolerated for a considerable time, and that up to a certain moment it may be quite possible to relieve the condition by the means just described? As already stated, the virus, coming from the ear in the first instance, is in certain cases mainly saprogenic, while in others it is intensely pathogenic. It remains for the future to discover means of distinguishing these varieties clinically from one another, so that we may know which cases to leave alone and which to treat by operation.

There remain now to be considered the modes of operating upon *definite abscesses in the brain and cerebellum*. I have elsewhere discussed this subject before, *àpropos* of two cases of my own, in both of which the operations were successful; but I may perhaps be pardoned if for completeness' sake I here once more take up the subject briefly.

In the first place, in what part of the encephalon are we to expect such abscesses, dependent on ear disease, to be found, as a rule? Fortunately, we are not dependent upon special nervous symptoms for the solution of this question. Our post-mortem records, as we have seen, furnish us with clear and unmistakable evidence upon it. We have seen that either the whole or part of each abscess of the brain lay within the "dangerous area"—in other words, in or about the middle of the temporal lobe. The largest abscesses have extended beyond the limits of this circle both in front and behind, but those of moderate size have been found within it always.

Now if a collection in the temporo-sphenoidal lobe is to be opened, I still think, after increased experience and more extended study of recorded cases, that the best point to be selected is that which I proposed and employed in my first case two years ago. This point (figs. 1, p. 36, and 9, *c*, p. 62) was then defined as $1\frac{1}{4}$ inch behind that centre of the bony meatus of the ear, and $1\frac{1}{4}$ inch above Reid's base line (*a a*, fig. 9) of the skull, which runs from the lower

border of the orbit backwards through the centre of the meatus of the ear.

From this point the whole of the dangerous area can be commanded, and if the abscess should happen to lie much in front of it the drainage will be the more complete from the opening being dependent, as the patient lies more or less upon his back, with the head inclined towards the affected side. If, on the other hand, the abscess is more than usually posterior, the exploring

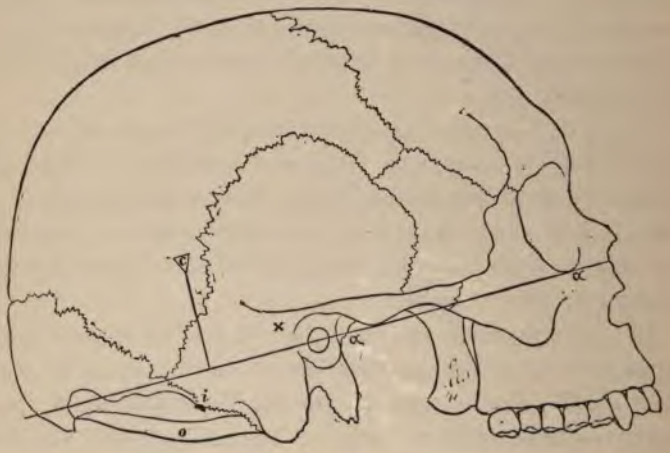

Fig. 9.

(Kindly lent by the publishers of the author's "Manual of Surgical Operations.")

From a photograph of a skull showing Reid's "base line," *a a*, the point **X** for opening the mastoid antrum, and the point *c* on the inferior angle of the parietal bone, $1\frac{1}{4}$ inch behind and above the centre of the meatus, at which temporal abscesses may best be reached.

needle can easily reach and empty it, as an anterior opening would not do. This spot has the further advantage that it is removed from the proximity of the meatus, and from the opening in the mastoid antrum previously made, which are usually both secreting more or less septic discharge. This point will be found to lie upon *the inferior anterior* angle of the parietal bone (figs. 9 and 10).

The farther away we get from this the better, obviously, the prospect of keeping our trephine opening aseptic. I know that a more anterior opening is advocated by surgeons whose opinions I cordially respect, but I venture to think that in the long run the view here put forward will be found to be correct, as it is based on pathological and clinical grounds.

As to the modes of opening the skull, I am now desirous of mentioning a simple modification which I adopted in a recent

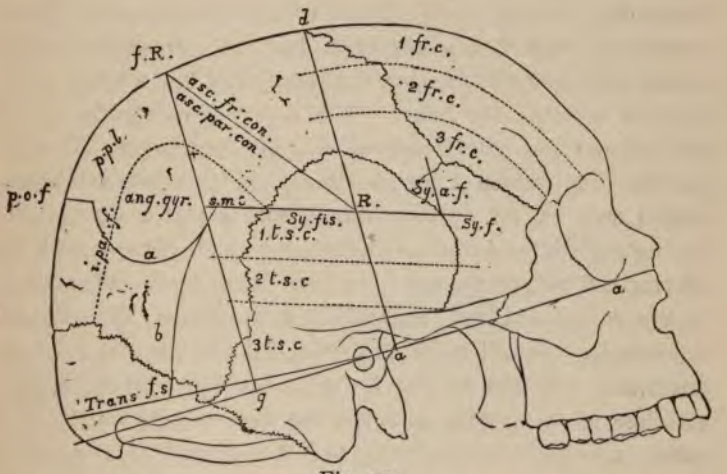

Fig. 10.

(Kindly lent by the publishers of the author's "Manual of Surgical Operations.")

From a photograph of a skull dissected by the author, showing the situations of the various sulci and convolutions. In fig. 1 the brain has been photographed *in situ* after removal of the bone in the same skull.

case, as it will, I think, simplify our procedure, and involve much less risk than the usual methods hitherto employed in such affections. I think I am correct in believing that, as a rule, it has hitherto been considered necessary to make a free incision in the soft parts down to the skull, and to open the latter by means of a rather large trephine. This was certainly the practice of several surgeons, including myself, in their first cases, and un-

doubtedly for some reasons it may be regarded as a suitable mode of procedure. But it occurred to me, some months ago that as the operation of opening the skull for this disease must in most cases be, in the first instance, purely exploratory, such an extensive procedure is quite unnecessary. In my last case of suspected temporal abscess, therefore, I operated as follows: After the usual cleansing of the scalp, the point indicated above as most suitable was marked on the latter, and over this a short, straight incision, three-quarters of an inch long, was made at one stroke down to the bone. The bleeding from this cut did not amount to more than a couple of drachms. The edges of the wound were now drawn apart sufficiently to allow a small ¼-inch trephine to reach the bone. With a few turns the latter was pierced, and the piece removed with the instrument. Through the hole thus formed I was able to explore the whole of the suspected area, as far as was desired, with a hollow needle, and had an abscess been reached, a drain-tube quite large enough for all practical purposes could have been introduced. The needle in this case was thrust forwards and backwards, upwards and downwards. It will be remembered that the bone at the spot selected is very thin, so that it did not interfere with the oblique direction of the needle, although the hole was so small. The entire operation, from beginning to end, only took eight minutes, and the loss of blood was quite insignificant. This modification appeared to me to contrast very favourably with the usual methods employed. In these much blood is lost, and a considerable amount of time is consumed in opening the skull. This may be in the highest degree injurious to a patient in the last stage of weakness, such as we have frequently to deal with. Of course, if, after reaching pus by the method just described, it were found that it could not be drained through the small trephine opening, nothing would be easier than to remove a larger disc of bone, including the first aperture. The soft parts would in such a case require to be divided more freely, and for this a horizontal cut to meet the first vertical one from behind would be most suitable.

The smaller opening in the skull would have the further advan-

tage, perhaps, of rendering hernia cerebri less likely, although in my own two cases, in which a large trephine was employed, this complication did not appear for a moment. This was probably due to the vigorous antisepsis employed throughout, which reduced the irritative hyperplasia to a minimum. We have ample evidence now before us to prove that whether the opening in the skull is large or small, hernia cerebri does not occur with antiseptic wounds of the brain, and that it is most marked where septic processes are most active. And that it is possible, in a case of abscess cerebri, to render the seat of operation perfectly aseptic, after evacuation of the pus, by careful irrigation and the guarded use of iodoform, these two cases, and many others besides, amply demonstrate.

Where the cerebellum has to be explored the simple method, just described ought to prove especially advantageous, for here the soft parts are thick and particularly vascular. In one of my own cases, operated on by the usual method of free incision for the application of a fair-sized trephine, I was startled by the amount of blood lost; and the time consumed in checking the bleeding was very considerable, factors in an operation which might prove very serious with a feeble patient.

Moreover, a small trephine is less liable than a large one to wound the lateral sinus. Finally, very little is gained by making the larger opening in the first instance, even supposing that inflammatory fluid is present in abundance. Should the latter be found, and should it not be evacuated by the small opening, it is very easy to use the larger trephine. If one were operating for meningitis the larger opening would require to be made probably in any case.

The spot (figs. 1 and 9, *o*) I have aimed at in any operations I have done for exploring the anterior portion of the cerebellum has in each case been, as nearly as possible, for the adult, 1½ inch behind the centre of the meatus and an inch below the base line, and this spot has always been found to be satisfactory. It is well below the horizontal part of the lateral sinus, and well behind the vertical part, while it gives easy access to all the anterior third of the lateral lobe, in which the pus, as we have seen, is invariably found.

As to drain-tubes, I still think that, whether for the brain or cerebellum, those made of silver are the most convenient. These are easily fashioned out of an old silver catheter, which, after being made nearly red-hot, is perfectly cleansed. Such tubes can be introduced with great ease and precision, and with a minimum of disturbance to the cerebral tissues, as will be admitted by any one who has used them. They can be given any curve necessary to enable them to reach the part of the brain desired. When it is time to reduce the depth to which they ought to reach a few layers of gauze are placed under the external flanges, or shorter tubes are used. The drainage with them is perfect, and if any granulations fungate into their lateral openings they are shaved off by the sharp edges of the latter as the tube is withdrawn, and thus the track is kept patent up to the last moment. The introduction of these tubes appears not to give rise to the slightest pain, and I have frequently discussed this point with my two patients while removing and replacing them. The same may be said of the irrigation of the abscess cavity, which should always be thorough but not forcible.

The most suitable dressings for these cases, in which foul pus has to be removed from the interior of the skull, are the various antiseptic gauzes, preferably that prepared with sal alembroth. This should be laid on in large quantity after being moistened, and should be covered by a piece of oiled silk or hat lining. It then becomes a species of fomentation, into which the pus escapes easily. In irrigating an abscess cavity probably the best antiseptic fluid is boracic acid solution. But whatever fluid is used, the danger of breaking down plastic adhesions round the trephine opening must not be lost sight of, and the whole procedure must be conducted with the utmost gentleness. In my own cases a very fine-pointed syringe has been introduced into the abscess cavity through the drain-opening twice or thrice a day, and the solution has been injected steadily until it ran out clean. No ill effect has ever been noted from filling the abscess cavity with the boracic solution. The latter has been selected for its unirritating properties, but probably other antiseptic fluids might prove equally *suitable. I have* been much struck by the almost immediate

cessation of discharge from these collections in the brain after the first dressing. The cavity appeared inclined to collapse and heal very rapidly.

It now only remains for us to review briefly the achievements of surgery in this field up to the present.

Of successful *operations upon the mastoid bone* for mischief in or about the sulcus lateralis we have now so many long series that it is not necessary to enumerate them. Considering the gravity ot the condition, the percentage of recoveries has been large. Such cases, as I have said, have occurred in my own practice, and have given very good results.

Cases of *subdural abscess*, both of the middle and posterior fossæ of the skull, have also been recorded in considerable number where life has been saved by operation.

Of removal of bone for *phlebitis of the lateral sinus* we need only say that enough successful cases have been placed on record to show that patients suffering from this condition should never be left to die without an attempt being made to reduce the amount of septic infection by operation and clearing away the damaged bone around the sulcus lateralis. Some very encouraging cases of interference for this condition have occurred comparatively recently at University College and elsewhere.

Typical cases ot *sub-acute pyæmia* with secondary deposits have also recovered after operations for cleansing the original seat of infection, and apparently as the result of this interference. Some of the cases of this kind have appeared desperate at the moment of operation, but the recovery has been rapid and complete nevertheless.

Instances also of *septic thrombosis* of the lateral sinus have been dealt with successfully. The broken-down clot has been reached by removal of bone, has been washed out, and thus an inevitably fatal result has been averted. Such results are still, of course, uncommon, but probably because we have not been bold enough in attacking this condition when it has been recognised or suspected. A case of this kind has, I believe, been successfully operated on at St. Thomas's Hospital within the last few weeks.

Meningitis, which perhaps, of all the conditions enumerated,

offers the greatest difficulties to the surgeon, has also been dealt with by operation with the very best results. I believe the first case of the kind occurred in my own practice. The case has already been reported in detail in the *British Medical Journal* for 1888, and I need not do more than allude very briefly to it here. The diagnosis was built up upon a study of the initial lesion in the ear, followed by close attention to the clinical symptoms. At last, when the patient was apparently moribund with deep coma, incontinence of fæces and urine, a pulse of 36, and more or less complete hemiplegia, I operated, on the hypothesis that he had a collection of meningitic fluid in or about the fissure of Sylvius. A thin, turbid, inodorous fluid was evacuated from this space, and recovery was rapid and complete. Of course this collection might have been primarily an abscess of the temporal lobe, and not a simple meningitic effusion. As the patient is now in good health and present here to-day, we have no means of absolutely demonstrating the exact nature of the inflammatory lesion. But from the course and nature of the symptoms, and from the appearance of the brain when I had exposed it in two separate spots freely, and from subsequent operations of exactly the same kind on the dead body, I am satisfied that the inflammatory fluid which I let out of this man's brain was pent up in the deeper part of the Sylvian fissure, in the space over the island of Reil (fig. 2, 1 2 3 4 5) and under the operculum (fig. 3, 2 and 3), and that my diagnosis of a simple meningeal collection was correct.

I show you here, sir, a photograph (fig. 11) of the patient's head, marked with the "base-line" (*a a*), the lines for the fissures of Sylvius and Rolando, and with circles indicating the spots at which the brain was exposed and punctured. The upper two circles (1 and 2) united formed an oval opening in the skull over the fissure of Rolando, at the situation of the arm centres. Punctures downwards and inwards here tapped the space (fig. 3, 2) over the island of Reil, and then the third or lower opening (3) enabled me to drain this space with a silver tube passing through the superior temporal convolution. The lower circle shows where this opening was made. I ought to say that *when I operated I* thought the meningitic collection would be

found on the superficial aspect of the ascending frontal and parietal convolutions, as well as in the Sylvian fissure, but that, in fact, it was on their deeper aspect, where they overhang the island of Reil.

So far as published records go, this appears to be the first case

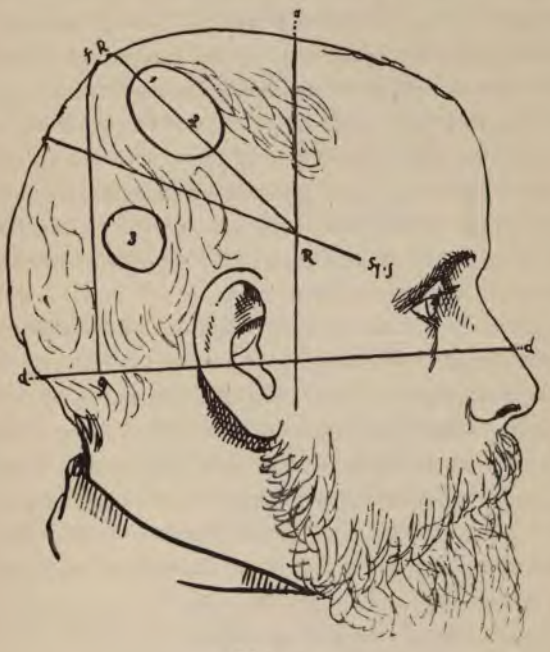

Fig. 11.

From a photograph of the patient's head. *a a*, "base line." *Syf.*, Fissure of Sylvius. *f.R. R.*, Fissure of Rolando. 1 and 2, first trephine openings over the face and arm centres (fig. 7, 6 and 7, *a b c d*). 3, the lower trephine opening made dependent for drainage.

in which an attempt has been made to deal with localised meningitis by operation. That it should have been successful under such very unfavourable circumstances is peculiarly gratifying.

Since Mr. Hulke's suggestion and bold attempt to relieve the brain of abscesses due to ear disease many successful operations of the kind have been done. One must regret that Mr. Hulke,

like many a pioneer whose work has led up to great results, was not then successful in his own cases. But it was his operations, no doubt, which commenced a new era in this field of surgery. I believe I have been privileged to perform the first successful operation for abscess of the brain due to ear disease yet recorded. This was in conjunction with Dr. Gowers, in the case of a patient present here to-day; and as it has already been described in the *British Medical Journal* in detail, I need not relate it in full. It was a simple case of temporo-sphenoidal abscess covered by healthy brain tissue, and when this was penetrated to the depth of half an inch fœtid gas and pus were evacuated with perfect recovery. This photograph (fig. 1, *c*) indicates the exact position of my puncture on the middle temporal convolution. It is marked with a small triangle of white paper, one inch and a quarter behind and above the meatus of the ear.

Mr. Caird, of Edinburgh, then followed with a second case, also successful, and Dr. Macewen with six or seven more, which he has fully described. Later, Mr. Horsley had a successful case of the same kind, and Mr. Jordan Lloyd another; and I have little doubt that others have been added since, of which we have not yet heard. I have not included in this list two cases operated on in Germany, one by Schondorff, the other by Schede, because I believe they come under a different category from our cases. In one a large and old sinus led down almost to the abscess, through the bone, and this had only to be enlarged in order to evacuate the cerebral collection. Of the other I will only say that Von Bergmann throws some doubt upon the conclusion that it was a true cerebral abscess, and suggests that the evidence is rather in favour of its having been simply a subdural collection of pus of large size.

At all events, we have here a collection of some twelve or fifteen cases where the brain has been relieved of inflammatory collections of fluid with the best results, in spite of impending death.

The only case in which the cerebellum has been successfully operated on for abscess due to ear disease has been already *alluded to as having* been treated so within the last few weeks.

This has fallen to the lot of Dr. Macewen, who has done so much other brilliant work in cerebral surgery, and no doubt the details will soon be before us.

In concluding this inquiry into the pathology, clinical history, and surgical treatment of the complications of suppurative disease of the temporal bone, I should like to say once more that throughout I have sought for facts rather than opinions, and have endeavoured to collect these from the most trustworthy sources. It was impossible, however, altogether to refrain from hypothesis, but in this I earnestly hope that due caution has been observed, and that the deductions drawn may not prove in any way dangerous. The whole subject is fraught with difficulty, and having experienced its intricacy, my aim has been to indicate and suggest those lines of inquiry which might in the future lead us most surely to the truth we seek, as well as to furnish those facts already ascertained. While searching for more light it must encourage us to know that our labours, even as far as they have gone, have yielded good fruit. Already our grand prerogative of giving back life and relieving suffering has been extended to a new class of unfortunates whose prospects, until quite recently, were hopeless. And should our inquiry result in the saving of only one more human life, or merely the alleviation of one single pang, surely the labour of conducting it will be overpaid a thousand-fold.

But whatever else we may have learned in our study of this subject, I trust we may never lose sight of the fact that operative surgery in its relation to these diseases, however encouraging its present results, is nothing but a "stop-gap," and that our first aim and endeavour should be to extend the knowledge of the origin and nature of these affections so widely that an intelligent and vigorous prophylaxis may become the rule at an early stage of the initial lesion, until these complications themselves have become a thing of the past.

To thank you, sir, and my audience, for the courteous and patient attention with which my efforts to interest have been received is my last and pleasantest duty.

I have, finally, the privilege of introducing to your notice two

of my patients already alluded to in dealing with cerebral suppuration, both saved from impending death by operation on their brains. The younger man was the first successful case of operation for cerebral abscess due to ear disease in this country, and probably anywhere; the elder one the first for localised meningitis ever performed, as far as I can tell.

My other patients, illustrating successful operative measures for subdural abscess, for phlebitis of the lateral sinus, and for pyæmic absorption, would no doubt interest you, but I am unable to find them at the present moment.

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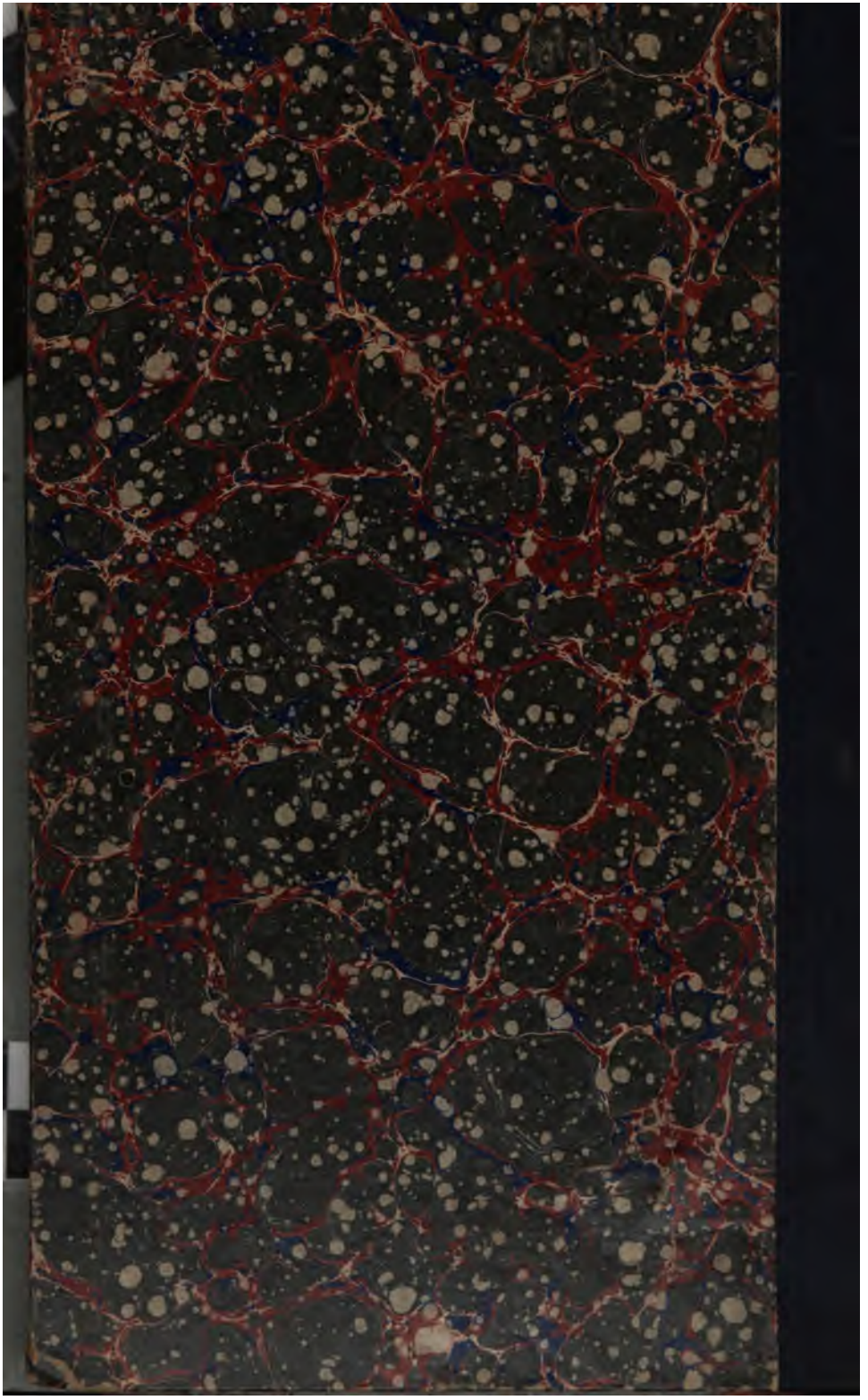
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